Gov. Ex. 163

2nd instructions: does that mean the letter A signifies it.?

A-TEST

Name: R. Breckman **Date:** 7/13/21

Directions: I am going to read you a long series of letters. Whenever you hear the letter A, please tap on the desk, but do not tap for any other letter. Any questions?

Administration and Scoring: Read the following letter list in a normal tone at the rate of one letter every 2 seconds (or every other second). Errors of commission and omission are counted as errors. The cutoff is >2 errors of any type.

Test Items:

Cross out error 1st trial

Circle error 2nd trial

L T P E A O A I C T D A L A A

A N I A B F S A M R Z E O A D

P A K L A U C J T O E A B A A

Z Y F M U S A late H E V A A R A T

#2 error 5

#1 **Errors:** 5

References:

(What did I ask you to do?) "It would all take place + be set up. The subsidiary other than that we wouldn't worry about it which was dealing w issues w the lawsuit"

Chafetz, M.D. (2012). The A-Test: A symptom validity indicator embedded within a mental status examination for social security disability. *Applied Neuropsychology, 19*, 121-126.

Chafetz, M.D., & Biondolillo, A. (2012). Validity issues in Atkins death cases. *The Clinical Neuropsychologist, 26*, 1358-1376.

GOVERNMENT EXHIBIT 4:21-CR-09-GCH No. 163

GOVERNMENT EXHIBIT 163

(What was I doing?) I think you was doing to make sure everything was done properly

(Did I read a series of letters?) yes

(What did I ask you to do?) Try to remember them

Repeated instructions

What am I going to do? what are you need to do i PC set up and send email for whatever you want

what are you here i me? our work is bldg software packages that are operated by people who are not top tier

who am I? a consultant charged by my profs i teaching more about the software and how it works and to bring prospects and suggest customers to U

what is my profession? a doctor who deals i 3rd party firms to help firms become more productive

Gov. Ex. 164

Green's Medical Symptom Validity Test

PATIENT: Brockman2, Robert
GENDER: Male
TEST DATE: Oct 2, 2021

EDUCATION: College Degree
DOB: [redacted]1941
TEST VERSION: English (Standard)

Patient Scores

	IR	DR	CNS	PA	FR
MSVT:	**90.0** (Pass)	**65.0** (Fail)	**75.0** (Fail)	**60.0**	**15.0**

Comparative Groups

	IR	DR	CNS	PA	FR
Patients with advanced dementia, German/oral MSVT,Brockhaus (N=14)					
Mean:	72.0	72.0	74.0	33.0	11.0
Standard Deviation:	19.0	18.0	15.0	23.0	12.0
Patient Z-Score:	0.9	-0.4	0.1	1.2	0.3
Patients with early dementia, German/oral MSVT,Brockhaus (N=48)					
Mean:	88.0	89.0	84.0	57.0	33.0
Standard Deviation:	13.0	13.0	13.0	26.0	22.0
Patient Z-Score:	0.2	-1.8	-0.7	0.1	-0.8

This person **failed** the effort subtests.
When asked if a full effort was made on this test, the subject said, "**Yes.**"
Self reported assessment of effort is **inconsistent** with performance.

Green's Medical Symptom Validity Test

PATIENT: Brockman2, Robert
GENDER: Male
TEST DATE: Oct 2, 2021

EDUCATION: College Degree
DOB: [redacted] 1941
TEST VERSION: English (Standard)

Patient Scores

	IR	DR	CNS	PA	FR
MSVT:	**90.0** (Pass)	**65.0** (Fail)	**75.0** (Fail)	**60.0**	**15.0**

WARNING: The above statements about failure and discrepancy with self-report apply only on the assumption that dementia is not present and that the person does not have a degree of cognitive impairment equivalent to dementia. The examiner should be familiar with the difference between the MSVT profile typically found in known simulators and most cases failing MSVT clinically, as opposed to the typical dementia profile. It is important to read the MSVT test manual sections on interpreting MSVT results, including Appendix B on patterns of MSVT scores consistent with dementia.

Implausible patterns of scores on MSVT can be produced by anyone not making a full and consistent effort across subtests. Having dementia does not guarantee valid test data. However, most patients with dementia score much higher on the easy MSVT subtests than on the harder subtests. That is because the first two subtests are objectively very easy and they are much easier than the last two subtests.

The mean of the easy MSVT subtests (IR, DR ,CNS) in 30 dementia patients from Dr. Brockhaus was 85% (SD=11), whereas the mean of the harder subtests (PA and FR) was only 40% (SD=21). The mean difference between the easy and hard subtests was 45% (SD=20). Please pay careful attention to the mean scores and SD's on MSVT subtests in early and advanced dementia patients and in simulator groups. Note especially the ratio of IR to FR scores, which is about 7:1 in cases of advanced dementia. Compare the scores from various groups with scores from the single case under examination. Look especially for implausible profiles on MSVT, suggesting unreliable data.

Implausible profiles on the MSVT are of many types. Some of the more common implausible profiles, which imply unreliable data due to poor and/or inconsistent effort, are identifiable by the following score patterns, in which both criteria A and B are met. Criterion A) IR, DR or CNS are at or below 85% and Criterion B) Any of the following are met: 1) The mean of the easy MSVT subtests (IR, DR ,CNS in % correct) is not at least 20 points higher than the mean of the harder subtests (PA and FR in % correct); 2) PA is less than or equal to FR; 3) PA-FR >=50; 4) IR-DR >= 15; 5) IR or DR <=FR

Green's Medical Symptom Validity Test

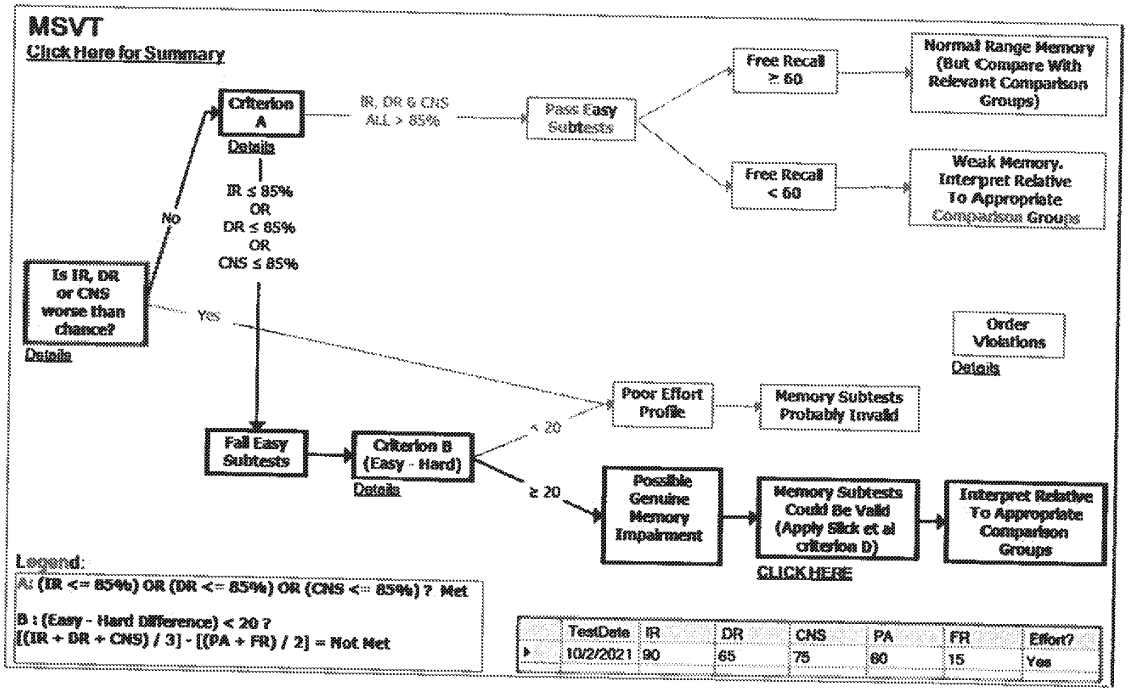
PATIENT: Brockman2, Robert
GENDER: Male
TEST DATE: Oct 2, 2021

EDUCATION: College Degree
DOB: [redacted] 1941
TEST VERSION: English (Standard)

Patient Scores

	IR	DR	CNS	PA	FR
MSVT:	**90.0** (Pass)	**65.0** (Fail)	**75.0** (Fail)	**60.0**	**15.0**

If IR, DR or CNS are 85% or lower, it is possible that the data are valid, if it is clinically plausible that the person has dementia or very severe impairment equivalent to dementia and if all of the following criteria are met: (a) at least a 20-point advantage of the mean of (IR, DR and CNS) over the mean of (PA and FR); (b) a higher score on PA than on FR and (c) a higher score on IR and DR than on FR. MSVT scores meeting these criteria might be consistent with dementia and good effort. This hypothesis would need to be confirmed by independent data, including clinical observations and, ideally, scores from the Word Memory Test for Windows (Green, 2003), the NV-MSVT (Green, 2006) and the Memory Complaints Inventory for Windows (Green, 1997-2004).



Patient: BROCKMAN2, ROBERT

MSVT Test Results: BROCKMAN2, ROBERT
Administration Date: 10/2/2021

IR: 90
DR: 65
CNS: 75
PA: 60
FR: 15

Summary:

This person has scored at or below the cut-offs on IR, DR &/or CNS, which can suggest poor effort and unreliable test scores. However, in this case, criterion B is not met because the easy-hard difference is at least 20 points. This profile can be found in people with genuine severe memory impairment, similar to that seen in people with dementia. This type of profile was also reported in people with severe dementia in the study of Singhal et al (2009) entitled "High specificity of the MSVT in people with very severe memory impairment" (in press, Archives of Clinical Neuropsychology).

Either passing criterion A or meeting criterion A but not meeting B was found in 95% of cases or more in several dementia samples. That is, meeting both A (low IR, DR & CNS) and B (easy-hard difference too small) has high specificity in genuinely severely impaired people. However, the sensitivity of this combination in known simulators is relatively low. That is, some simulators can mimic a possible dementia profile.

If the person meets criterion A but not B, they have a "possible dementia profile" or GMIP (genuine memory impairment profile) on the MSVT but the possibility of a false negative for poor effort should be considered. The assumption of good effort but severe impairment should only be made if the person's clinical presentation, history and diagnosis are consistent with the level of memory impairment found in people with dementia.

Are you prepared to diagnose dementia or an equivalent condition (e.g. severe memory deficits from bilateral hippocampal damage)? If not, poor effort probably needs to be concluded. If you are entertaining the possibility of dementia or an equivalent disease, sufficient to cause low scores on IR, DR or CNS, this could have very serious implications for the person. For example the person might need guardianship and trusteeship; medical facilities and accommodation for dementia

patients may need to be provided; the person (and family) may be told that work is not possible and disability benefits may be made available. These steps would be inappropriate and potentially harmful to the client and his/her family if the person were actually feigning impairment.

The risk of a false negative for poor effort can be reduced and sensitivity to poor effort can be increased without significantly reducing specificity. This can be achieved by testing the person with the WMT or the NV-MSVT and preferably both, in addition to the MSVT. For example, see the study of people with severe dementia given both the MSVT and NV-MSVT in the study of Singhal et al (2009) entitled "High specificity of the MSVT in people with very severe memory impairment" (in press, Archives of Clinical Neuropsychology).

"Is any recognition test score (IR, DR or CNS) worse than chance?" No (see combinations below)

For MSVT IR, DR and CNS, there are 20 items each. Each response has a 50-50 probability of being right by chance alone. A score of 6/20 (i.e. 30%) or lower is significantly worse than chance, if we set the cut-off at ($p \leq .06$). That is, it would occur by chance alone less than 6 times out of 100 ($p<.058$).

"Is any combination worse than chance?" No

For MSVT IR and DR combined, there are 40 items. Each response has a 50-50 probability of being right by chance alone. A score of 14/40 (i.e. 35%) or lower is significantly worse than chance. That is, it would occur by chance alone less than 5 times out of 100 ($p<.05$).

The sum of the raw IR and DR scores (i.e. number correct), expressed as a percentage of 40 (total responses) is 77.5%. This is above the chance level.

Criterion A: "Is IR, DR or CNS 85% or lower?" Yes

This means that this person has scored below the cut-off on at least one of the easy

MSVT subtests or on consistency between these subtests (CNS). Such scores are rare in adults or children with a grade 3 reading level or higher, who try their best to do well on the MSVT. The scores should be interpreted relative to appropriate comparison groups, using the REPORTING button in the MSVT program and selecting REPORT BUILDER and, later, CHART BUILDER. Care should be taken in interpreting any IR, DR or CNS score of 85% or lower. Such scores are very rare, except in adults with dementia or people with a FSIQ below 70.

The severe degree of such memory impairment can be appreciated by considering the MSVT scores reported by Dominic Carone in his paper on children with severe traumatic brain injuries or developmental disabilities. (Carone, D. Children with moderate/severe brain damage/dysfunction outperform adults with mild to no brain damage on the Medical Symptom Validity Test. Brain Injury, 2008; 22, 12, 960-971.) All children in the latter study who tried to pass the MSVT IR and DR subtests did so. They rated them as being extremely easy. Yet a substantial number of adults with mild TBI were reported to fail the same subtests. Those who failed the easy MSVT subtests rated the IR and DR subtests as "moderately difficult".

Scores below the cut-offs on IR, DR or CNS can arise from one of two underlying causes. Either (a) the person is capable of scoring at a higher level and the observed score is not reflective of true ability (poor effort, for short) or (b) the observed score represents the person's actual capability when motivated to do well *and* the person has severe verbal memory impairment or some other severe cognitive deficit, similar to that seen in some cases with dementia. In order to assist in discrimination between these two options, we calculate the difference between the mean scores on the "easy" (IR, DR & CNS) and "hard" subtests (MC, PA & FR).

Criterion B: "Is the easy-hard difference less than 20?" No

Criterion B is not met. In this person, the "easy-hard difference" (mean IR, DR & CNS – mean PA & FR) is 39.17 points. This difference is 20 or more and such differences are found in people with dementia or other very severe impairment. Clinical correlation is required to interpret such an easy-hard difference score as being indicative of severe cognitive deficit. To draw this conclusion, the person must have a clinical diagnosis, history and presentation consistent with very severe cognitive impairment, such as dementia. If the person does not have a clinical diagnosis, history and presentation consistent with very severe cognitive

impairment, such as dementia, then it must be concluded that the test results probably underestimate the person's actual capabilities and are invalid.

Gov. Ex. 165

APPLIED NEUROPSYCHOLOGY: ADULT
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Routledge
Taylor & Francis Group

Use of the Rey 15-Item Test as a performance validity test in an elderly population

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ABSTRACT

The objective of this study was to determine the effectiveness of the Rey 15-Item Test in elderly individuals with and without cognitive impairment and to develop new indices to function with this population, if needed. The sample consisted of 185 individuals referred for outpatient neuropsychological evaluation. All were clinically evaluated and administered the Rey 15-Item Test (RFIT) with recognition as part of this procedure. Significant differences were present between those passing and failing the RFIT on referral question; working status; age; and diagnosis of cognitive impairment. Individuals age 60+ failed the test at rates in excess of 35%. Diagnosis also played a role with those with more severe cognitive diagnosis failing at higher rates; even in those with no diagnosis, however, 19% of elderly individuals did not produce a passing score. The extant cutoff scores commonly used with the RFIT produce unacceptably high false positive error rates to be a useful freestanding Performance Validity Test (PVT) with individuals above age 59. The introduction of a new combination score and use of rare scores and patterns of scores improves the psychometric properties of the RFIT when used with elderly adults. Cautious use of PVTs is warranted until they have been validated with aged populations.

KEYWORDS

Age; performance validity; psychometric properties; Rey 15-Item Test; specificity

Introduction

Per the 2010 census, approximately 13.0% of the population in the United States is 65 years of age and over, more than three times that of just over a century ago (U.S. Department of Commerce, U.S. Census Bureau, 2014). This is, by many definitions, considered elderly, although "elderly" has been defined as young as the age of 60 by some organizations (World Health Organization, 2013). The proportion of aging U.S. citizens is rising rapidly and anticipated to reach 20% of the total population by the year 2040 (U.S. Department of Health and Human Services, 2008). As neuropsychologists, evaluators are tasked with ensuring evaluees' performance validity as it is an established professional standard of practice (Bush et al., 2005; Heilbronner, Sweet, Morgan, Larrabee, & Millis, 2009). Yet, there is a minimal amount of published research on how elderly populations—who genuinely experience cognitive decline associated with age in various domains—perform on what are commonly viewed as "easy" tests, namely Performance Validity Tests (PVTs).

All patients can be motivated to underperform for reasons related to forensic matters, such as personal injury lawsuits or criminal charges. Older patients may also put forth suboptimal effort for reasons such as resistance to being assessed, fatigue, poor cognitive stamina, or not fully understanding the necessity and/or consequences of the evaluative outcome (Bortnik, Horner, & Bachman, 2013). As previously alluded to, however, the elderly may also produce what appear to be failing scores due to genuine declines in the underlying cognitive abilities necessary for successful performance, and these declines can be exacerbated in the face of additional cognitive decline caused by early neurodegenerative disease.

PVTs have only recently been considered in elderly patients, including those with mild cognitive impairment and dementia (Dean, Victor, Boone, Philpott, & Hess, 2009). In those studies that have been completed on PVTs in the elderly, results have been varied with outcomes tending toward poor specificity levels (Bortnik et al., 2013; Dean et al., 2009; Loring et al., 2016; Rudman, Oyebode, Jones, & Bentham, 2011). Even in studies not specifically looking at elderly populations, however, there has been the suggestion that older age can cause poorer specificity on some validity indicators (Reedy et al., 2013).

Most recently, several studies have been published regarding the functioning of Reliable Digit Span

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A portion of this data was presented at the National Academy of Neuropsychology Conference, 2016, in Seattle, Washingto

(RDS) in older adults with suspected cognitive problems and those with Mild Cognitive Impairment or early Alzheimer dementia (MCI; Loring et al., 2016; Zenisek, Millis, Banks, & Miller, 2016). These studies indicated that the commonly used cutoff for RDS has to be dropped by approximately 2 points or used in conjunction with other measures to produce an algorithm in order to maintain adequate specificity in the elderly who are clinically diagnosed with MCI or dementia. By making this alteration, it appears that RDS can be used with the elderly, with specificity of above 90% in those with no cognitive diagnosis, MCI, early Alzheimer dementia, and several other types of neurodegenerative disorders, with the possible exception of frontotemporal dementia (Loring et al., 2016; Zenisek et al., 2016).

Several other PVTs can be used with those with genuine memory impairment, such as Green's Word Memory Test, Medical Symptom Validity Test, and Non-Verbal Medical Symptom Validity Test (Green 2004, 2005, 2008). Again, however, this was after modification of these tests to include a Genuine Memory Impaired Profile (GMIP), to essentially make additional allowances/develop a performance algorithm for those who have genuine memory impairment, whether that be due to increasing age, frank dementia, or other neurological factors.

The Rey 15-Item Memorization Test (RFIT; Rey, 1964) and Recognition Trial (Boone, Salazar, Lu, Warner-Chacon, & Razani, 2002), is a commonly used PVT (Slick, Sherman, & Iverson, 1999). As of 2007, the RFIT was used at least on occasion by 74.5% of surveyed neuropsychologists (Sharland & Gfeller, 2007). A later survey cast further light on the use of the RFIT, with 24.1% of the sample using some version of the RFIT, and 7.9% specifying use of the RFIT + recognition trial (Martin, Schroeder, & Odland, 2015). Unfortunately, in the initial study establishing the recognition trial, those with suspected dementia were eliminated from the clinical group, which the authors stated both enhanced specificity values and also left the question of using the RFIT with recognition in those with dementia unanswered. Alternately, because of the older age of the control group vs. the suspect effort group, the authors concluded that the RFIT plus recognition could be used with "an unrestricted age range" (Boone et al., 2002, p. 571). This represents false logic since clinically, "controls" do not often present for neuropsychological evaluation, and the mean age of the clinical group was 41.

There have been other studies, however, that have attempted to evaluate the performance of the RFIT recall or RFIT plus recognition in individuals with dementia and/or a broader age range. Schretlen, Brandt, Krafft, and Van Gorp (1991) reported the RFIT had unacceptable specificity in dementia patients with the standard cutoff of <9. Nevertheless, their group of dementia patients consisted of a small sample of nine, six of which were Huntington's dementia patients. A more recent study specifically examined 214 dementia patients with 18 effort indices (calculated from 12 tests), among them the RFIT, and found unacceptably low specificity for the RFIT (Dean et al., 2009). In the above noted research, age ranged from 23–97 years old with a mean of 63.5 years, all patients (from two samples) were diagnosed with a form of dementia (e.g., frontotemporal, Alzheimer's, Lewy body, substance abuse, lupus, AIDS, TBI, etc.) and the researchers evaluated whether level (mild, moderate, or severe) or type of dementia affected performance on the 18 effort measures analyzed. The nature of their study, without a group of patients who did not meet the criteria for a dementia diagnosis, did not allow the researchers to examine how age or age in conjunction with other cognitive diagnoses might also affect performance on the RFIT. Dean et al. (2009) concluded that the cut-offs traditionally used on the RFIT (<9 for free recall or <20 for recall + recognition), among other measures, were unacceptable for those with a diagnosis of dementia to assess effort in neuropsychological evaluations.

Similarly, Bortnik et al. (2013) found, despite best effort, older adults with a dementia diagnosis were less likely to pass stand-alone or embedded effort measures during a neuropsychological evaluation, with false-positive error rates that exceeded acceptability. Their research showed the RFIT free recall and RFIT combined trial scores had 100% sensitivity, but only 28% specificity on the free recall trial and 0% specificity on the RFIT combined trial. Thus, they found this measure an unacceptable measure of performance effort in patients with dementia. Again, these researchers did not report findings of older adults who did not meet diagnostic criteria for dementia.

This current research examines use of the RFIT, predominantly the combination score with both free recall and recognition, in older adults with potential memory impairment. Extant cutoffs are inspected, and methods of modifying the RFIT to make it more suitable for use with older adults, such as had been done with other neuropsychological tests, are also examined.

Method

Participants

The sample consisted of 185 individuals referred for outpatient clinical neuropsychological evaluation at a

private practice in the Southeastern United States; this was after excluding any participants with potential medicolegal issues and those who did not receive the RFIT as part of the testing battery.

Materials and procedure

All evaluees were administered the Rey 15-Item Test (Rey, 1964) with recognition trial (Boone et al., 2002) as part of a clinical neuropsychological assessment. Since the evaluation was performed to answer clinical questions, participants did not receive a standardized test battery. As such, the RFIT was also not administered in the same order to all participants. All participants who were involved in this research signed an informed consent form for their clinical data to be maintained in a research database.

Data analysis plan

In addition to calculating descriptive statistics for the entire sample and those passing and failing the RFIT, comparisons were performed utilizing chi-square tests for categorical data and Mann-Whitney U tests for continuous data as most of the data were slightly skewed, stretching the limits of normality. Follow-up analyses were planned using Spearman correlations (one-tailed to follow up the initial results) to evaluate the strength of the relationships between variables, and logistic regression to determine their relative contributions to RFIT outcome. The contributions of age and diagnosis were examined separately to be of greater use to clinicians. In these cases, data were broken down categorically and analyzed using chi-square and Jonckheere's trend test (Jonckheere, 1954), as appropriate. Finally, variables were explored and new equations produced in order to maximize the clinical utility of the RFIT with elderly individuals.

Results

Descriptives

Of the overall sample, 55.1% were female with the remainder male. The sample was predominantly Caucasian (95%) and 4% African-American. Average age was 67.9 years (±15.4). Mean education was 14.2 years (±2.7). Age ranged from 16–89; although the focus of this paper is on older individuals' performances (as defined by the United Nations, 2013, as 60 or older), the younger participants were retained for comparison purposes. The majority were referred by their primary care physician or a neurologist (91.4% total) for memory complaints (84.9%). Most were retired (59.5%). After the clinical evaluation, 20% were deemed to not meet diagnostic criteria for any diagnosis related to cognitive functioning, whereas 43.2% met diagnostic criteria for Mild Neurocognitive Disorder (hereafter referred to as MCI [Mild Cognitive Impairment] for clarity) and 28.1% for Major Neurocognitive Disorder (MND) per DSM-5 diagnostic criteria (American Psychiatric Association, 2013). The remaining 8.6% met criteria for another diagnosis related to cognitive functioning, predominantly Attention Deficit Hyperactivity Disorder (ADHD).

Main analyses

Ensuring data validity

Although it was discussed above that there is a dearth of information on how various PVTs perform in an elderly population with potential dementia, given that RDS is now validated with this population, the sample's RDS values were examined to compare this sample to others with dementia and presumed good effort. Of the sample, 165/185 of the participants had received enough of the Digit Span subtest of the WAIS-IV in order to calculate RDS (i.e., digits forward and backward until discontinue rule was met). RDS was calculated as per Greiffenstein, Baker, and Gola (1994) for all participants. Of these, only 2.4% produced a RDS score of 5; no participant produced a score below 5 (See Table 1 for specificity at various RDS values). It should be noted, however, that the 20 participants who did not receive enough of the digit span subtest in order to calculate RDS may have had more severe cognitive impairment which led to the clinical decision to not administer the test; if these participants had received this test, the specificity value may be lower. This hypothesis was supported by the fact that 17/20 of the participants who had missing RDS data were diagnosed with dementia. Regardless, the values seen in this sample are very much in line with those from previous samples of individuals evaluated for potential dementia (Loring et al., 2016; Zenisek et al., 2016). As such, all participants were retained for subsequent analyses.

Table 1. Percentage of below criterion performance.

	RDS ≤5	RDS ≤6	RDS ≤7
Full sample	2.2	8.7	20.6
No diagnosis	2.8	5.6	11.2
LD/Other	0	0	18.8
MCI	0	11.5	24.3
Dementia	8.6	14.3	34.3

Note. Sample size was $n = 165$. RDS = Reliable Digit Span. Diagnoses: LD = learning disorder; MCI = mild cognitive impairment; MND = major neurocognitive disorder.

Failure rate on the RFIT

In the overall sample, 55% produced a passing score on the RFIT (defined as a score of ≥20 on recall + recognition as per Boone et al., 2002), and 45% failing. Chi-square analyses indicated the two groups did not differ in gender, ethnicity, or referral source. They did, however, differ in referral question, retirement status, and cognitive diagnosis, with those failing the RFIT being more likely referred for memory problems, retired, and having a clinical diagnosis of MND. See Table 2 for these percentages and statistics. Regarding the continuous variables, those passing and failing the RFIT were no different when it came to estimated premorbid IQ (IQ estimation methods were combined and consisted of the Hopkins Adult Reading Test, North American Adult Reading Test, and Test of Premorbid Functioning Standard Score; Schretlen et al., 2009; Blair & Spreen, 1989; and Pearson, 2009; respectively). Alternately, there were significant differences between the two groups on age and current Full Scale Intelligence Quotient (FSIQ) as well as education level. See Table 3 for these values. In order to further provide some characterization of the two groups' cognitive abilities, also see Table 3 for descriptive statistics for various neuropsychological tests administered. There were differences between scores on all tests between the groups that had passed and failed the RFIT.

Table 2. Categorical variables by group.

Variable	Pass ($n = 101$) %	Fail ($n = 84$) %	Test, p value
Gender			
Male	50.6	49.4	$\chi^2 = .968$, $p = .325$
Female	57.8	42.2	
Ethnicity			
Caucasian	54.9	45.1	$\chi^2 = 2.611$, $p = .456$
African-American	62.5	37.5	
Referral source			
PCP	55.4	44.6	$\chi^2 = 5.921$, $p = .432$
Neurologist	49.4	50.6	
Other	25.0	75.0	
Diagnosis			
None	86.5	13.5	$\chi^2 = 64.912$, $p < .001$
LD/Other	81.3	18.8	
MCI	63.8	36.3	
MND	9.6	90.4	
Retired			
Yes	38.2	61.8	$\chi^2 = 37.864$, $p < .001$
No	90.2	9.8	
Other	64.7	35.3	
Referral Question			
Memory	48.4	51.6	$\chi^2 = 16.991$, $p = .001$
ADHD	100	0	
DVR	100	0	
Other Cog. Eval.	54.6	45.4	

Note. PCP = primary care provider; LD = learning disorder; MCI = mild cognitive impairment; MND = major neurocognitive disorder; ADHD = attention deficit hyperactivity disorder; DVR = Division of Vocational Rehabilitation.

Table 3. Continuous variables by group.

Variable	Pass ($n = 101$) M (SD)	Fail ($n = 84$) M (SD)	Test, p value
Age	61.9 (17.1)	75.2 (8.7)	$U = 2208.0$, $p < .001$
Education	14.7 (2.6)	13.6 (2.6)	$U = 3241.0$, $p = .005$
Estimated FSIQ[a]	102.4 (12.1)	100.7 (12.1)	$U = 3403.0$, $p = .502$
Current FSIQ[b]	102.3 (14.2)	95.1 (15.4)	$U = 600.0$, $p = .027$
MMSE[c]	27.4 (1.8)	24.1 (4.0)	$U = 353.0$, $p < .001$
COWA Animals	16.8 (5.0)	11.5 (4.3)	$U = 1434.0$, $p < .001$
COWA FAS	36.4 (11.8)	29.7 (11.1)	$U = 2390.5$, $p < .001$
BNT	53.6 (7.0)	48.6 (7.6)	$U = 1724.0$, $p < .001$
TMTA (seconds)	41.7 (20.0)	73.7 (55.0)	$U = 1862.0$, $p < .001$
TMTB (seconds)	104.4 (57.9)	204.3 (117.3)	$U = 997.0$, $p < .001$
CVLT-SF Imed. Recall	25.2 (4.5)	20.7 (4.3)	$U = 874.0$, $p < .001$
CVLT-SF Delayed Recall	5.6 (2.2)	2.6 (2.7)	$U = 828.5$, $p < .001$
RCFT Imed. Recall	14.1 (6.3)	5.5 (4.9)	$U = 632.0$, $p < .001$
RCFT Delayed Recall	13.6 (5.9)	4.9 (4.3)	$U = 503.0$, $p < .001$

Note. FSIQ = Full scale intelligence quotient; MMSE = Mini Mental Status Exam; COWA = Controlled Oral Word Association; BNT = Boston Naming Test; TMT = Trail Making Test; CVLT-SF = California Verbal Learning Test-II, Short Form; RCFT = Rey Complex Figure Test. [a]Most but not all participants had a measure of IQ estimate; $n = 171$. [b]Fewer individuals had a FSIQ in the "Fail" group than the "Pass" group, likely due to a full WAIS-IV not being administered because of their advanced age and propensity to have greater cognitive impairment. Sample size was $n = 63$ in the "Pass" group and $n = 27$ in the "Fail" group. [c]Sample size continued to vary by test given that this was a clinical sample and not all tests were administered to all participants. Sample size for the neuropsychological tests ranged from a low of 37/101 to a high of 92/101 in the "Pass" group and from 43/84 to 80/84 in the "Fail" group.

The strength of the relationships between demographic variables and RFIT score was also explored. RFIT score as a continuous variable had the strongest correlations with age ($r_s = -.530$, $p < .001$) and collapsed diagnosis (as listed in order in Table 2; $r_s = -.612$, $p < .001$). There were also significant correlations with education ($r_s = .181$, $p = .007$) and premorbid IQ estimate ($r_s = .172$, $p = .012$), but they were substantially weaker. These results were then confirmed by using logistic regression to determine if these variables had a significant influence in group classification. Several models were explored; in each model, age and diagnosis were significant predictors. Estimated premorbid FSIQ did not make a significant contribution. Somewhat expectedly, when both IQ and education were entered into models, only one or the other were retained. It was therefore decided to retain education as a predictor, as this variable was available for every participant, whereas IQ was only available for about half of the sample. The final model containing age, collapsed diagnosis, and education was highly significant and explained between 41.9 and 56.0% of the variance. See Table 4 for these values.

How was the RFIT affected by age and diagnosis?

From a clinical perspective, it is important to understand how these psychometric properties apply to the

Table 4. Logistic regression values for predicting pass/fail on the Rey 15-Item Test.

Variable	β	SE	Wald	df	p	Odds Ratio	95% CI, Lower	95% CI, Upper
Collapsed Diagnosis	–	–	31.243	3	<.001	–	–	–
Collapsed Diagnosis (1)	−1.626	.982	2.742	1	.098	.197	.029	1.348
Collapsed Diagnosis (2)	−1.368	.595	5.292	1	.021	.255	.079	.817
Collapsed Diagnosis (3)	−3.945	.738	28.537	1	<.001	.019	.005	.082
Age	−.073	.020	12.893	1	<.001	.930	.894	.968
Education	.272	.082	10.925	1	.001	1.312	1.117	1.542
Constant	3.187	1.814	3.088	1	.079	24.221	–	–

Note. Fail was coded as "0" and Pass as "1." For Collapsed Diagnosis, 0 = No diagnosis; 1 = LD/Other; 2 = MCI; and 3 = MND. LD = Learning disorder; MCI = mild cognitive impairment; MND = major neurocognitive disorder.

everyday use of the RFIT with older adults. As such, the data were then collapsed into age brackets, starting with all of those under age 40, and proceeding by ten year increments (40–49, 50–59, etc.). Although the maximum RFIT Combination Score was 30 for all but the oldest age group (80–89, max. = 27), the minimum score demonstrated a nearly-linear decline as age increased, starting at 24 in those under 40 and declining to 0 for those in their 80s. The mean also showed a nearly-linear decline, although with some variability likely due to differences in sample size between groups. Similarly, those under 40 had a 100% pass rate on the test, while those in the oldest group had only a 38.5% pass rate (and therefore a false positive rate of 61.5%). These values can be seen in Table 5.

Due to the similarities in performance amongst those ages 16–59 and ages 70+, these age groups were further collapsed to form a ternary age classification (ages 16–59, 60–69, and 70+). This demonstrated that this test, up to age 59, has an acceptable specificity (.894). This changes, however, in those age 60–69 (.645) and 70+ (.364; as demonstrated in Table 5). The differences in failure rates between these groups were highly significant ($\chi^2 = 38.4$, $p < .001$). The differences held when the RFIT combination score was examined across groups (Standardized $S = -7.8$, $p < .001$).

Regarding diagnosis, again those in nearly every diagnostic group had a maximum RFIT combination score of 30; the exception to this was the MND group (max = 27). The minimum scores, however, were somewhat more variable. The mean score demonstrated a negative relationship with diagnosis (i.e., the mean score decreased as diagnostic severity increased). The percentage of those producing passing scores in each group had a similarly negative relationship. These values can be seen in Table 6. It should be noted that even those in the "no diagnosis" group had a somewhat higher than expected failure rate on this test; this was confounded by their age, as they had a mean age of 64 years old, whereas those in the "LD/Other" group were actually younger with a mean age of 46. Those with MCI were 69 years old, and those with MND 77 years old; this again illustrates the importance of age on test performance.

To further clarify this relationship, those with no cognitive diagnosis were examined separately from the rest of the sample. In this group ($n = 37$), 93.8% individuals through age 59 produced a passing score on the RFIT. Among those ages 60 and over, however, 4/21 (19%) did not produce a passing score, which is an unacceptably high failure rate. As can be seen in Table 7, if the RFIT was to be used as a freestanding PVT in those with known (previously diagnosed) MCI or Dementia, both the Recall and Combination Scores would have to be reduced very substantially in order to maintain adequate specificity in this population.

Table 5. Age and RFIT scores/passing rate.

Age group (*n*)	Mean score (*SD*)	Min/Max	Passing %	Failing %
<40 (11)	28.5 (2.4)	24/30	100	0
40–49 (10)	26.1 (4.3)	18/30	90.0	10.0
50–59 (26)	25.5 (6.7)	3/30	84.6	15.4
60–69 (31)	21.3 (6.8)	6/30	64.5	35.5
70–79 (68)	15.8 (7.8)	1/30	35.3	64.7
80–89 (39)	15.9 (7.6)	0/27	54.6	45.4

Table 6. Diagnosis and RFIT scores/passing rate.

Diagnostic group (*n*)	Mean Score (*SD*)	Min/Max	Passing %	Failing %
No diagnosis (37)	24.5 (5.5)	3/30	86.5	13.5
LD/Other (16)	25.4 (5.9)	11/30	81.3	18.8
MCI (80)	21.4 (7.0)	0/30	63.8	36.3
MND (52)	10.9 (5.8)	1/27	9.6	90.4

Note. LD = Learning disorder; MCI = mild cognitive impairment; MND = major neurocognitive disorder.

Table 7. Specificity of RFIT variables by diagnostic group.

Diagnosis	RFIT Recall ≤	Specificity	RFIT Combination ≤	Specificity
No Diagnosis	8	.946	12	.946
	9	.892	17	.919
	11	.811	19	.865
MCI	5	.937	10	.912
	6	.887	11	.900
	7	.875	12	.887
MND	1	.981	3	.904
	3	.769	5	.865
	4	.750	6	.769
	5	.712	7	.673

Note. MCI = mild cognitive impairment; MND = major neurocognitive disorder.

Can the RFIT be adjusted to work with an elderly population?

Available RFIT variables were qualitatively examined to see if any adjustments could be made to the combination score to make it more amenable to use with an elderly population. Recall was deemed unlikely to be useful as "3" was a common score in this group, although it was noted that only 1.4% of those ages 60+ had a score less than 3. Scores on the recognition test were somewhat more promising, although given the very low basal for these, sensitivity in a population with incentive to under-perform may also be unacceptably low. Regardless, of those ages 60+, only 5.7% had <3 true positives on the recognition portion of the test, and only 8.6% had >5 false positives. The extant combination score would have to be dropped from <20 to <7 to maintain adequate specificity amongst those age 60+ (spec = .89). See Table 8 for corresponding sensitivity/specificity values for extant RFIT variables in those ages 60+.

Given the relatively poor performance of the variables presently used to assess validity, number of intrusions was also evaluated as this had been mentioned in another population as a potentially useful validity indicator ("drawing of an item not on the original stimulus card;" Lee et al., 2000). Intrusions were coded later in the data collection process, however; as a result, only a subsample of participants ($n = 103$) had this data. Number of intrusions was rarely above 3 among individuals age 60+, making this a potentially useful indicator. Specificity data for this variable is also in Table 8.

A new combination score including true positives and false positives from the recognition trial in conjunction with intrusions was also evaluated given that these variables all independently seemed to have somewhat better specificity than recall. This was calculated as (true positives—false positives—intrusions); this resulted in scores ranging from −18 to +15. Scores of <2 were uncommon, occurring in only 8.7% of the sample; all individuals producing scores <2, however, were ages 60+. The full range of values for this new combination score can be seen in Table 9.

Table 8. Specificity of RFIT variables in age 60+.

Value	Recall (≤)	True+ (≤)	False+ (>)	Combination Score (≤)	Intrusions (>)
0	.993	.971	.435	.993	.732
1	.986	.971	.609	.986	.831
2	.986	.942	.732	.986	.859
3	.898	.884	.833	.957	.930
4	.877	.848	.877	.957	.944
5	.841	.804	.913	.942	.958
6	.681	.710	.949	.891	.986
7	.659	.696	.978	.856	.986
8	.587	.659	.978	.833	.986

Table 9. Frequency of scores on new combination score variable.

Value	%	Cumulative %
<0	3.9	3.9
0	2.9	6.8
1	1.9	8.7
2	4.9	13.6
3	9.7	23.3
4	1.9	25.2
5+	74.8	–

Note. Due to including intrusions in this formula, which were not available for the entire sample, $n = 103$.

Pattern analysis of scores was also attempted to see if a GMIP-type pattern could be discerned. Recall versus recognition was examined, but quickly abandoned when it was found that 35.1% of the sample produced a higher score on recall than on recognition true positives. Alternately, the prevalence of the previously mentioned rare scores (recall <3, true positives <3, and false positives >5) were examined in combination to see if any of these combinations could be used as a pathognomonic marker of likely disingenuous performance. This was much more effective. Of the total sample, 0% of individuals produced a score of recall <3 in conjunction with either a score of true positives <3 or a false positive score >5; this indicates the prevalence of these scores if considered altogether (all 3 scores) was also 0%. On the other hand, there was a prevalence of 1.1% if recall <3 and true positives <3 was considered, which is somewhat logical as these individuals seemed to be unwilling to endorse items across the board.

Discussion

The goal of this study was to determine the efficacy of the RFIT as a potential measure of performance validity amongst a clinical population of elderly individuals with heterogeneous diagnoses. Determining the effectiveness of PVTs in those ages 60+ is becoming increasingly important as lifespan increases and elderly individuals form a larger proportion of the population. Just as younger individuals may, the elderly can also be involved in criminal or civil litigation where effort on cognitive testing can be an important variable, although even screening for effort in routine clinical evaluations can provide important insights into test-taking approach. Unfortunately, given the aforementioned results, it does not appear the extant indices of the RFIT are effective tools to utilize for evaluating performance validity in elderly individuals.

Fortunately, however, there are some indicators that scores above or below certain cutoffs on portions of the RFIT occur only very rarely; linking these occurrences together leads to scores that are so rare they can be

considered pathognomonic, such as seen with certain performance patterns on the Dot Counting Test (Boone, Lu, & Herzberg, 2002). These findings make some logical sense as well as statistical sense; it does seem unlikely, for example, that an individual would get almost no correct answers for either recall or recognition but would make an excessive number of false positive identifications. Intrusions were also rare in this study, particularly a high number of intrusions. This remains a promising variable for further investigation.

Interestingly, pattern analysis involving recall > recognition was not a promising metric due to the number of individuals achieving a higher score on the recall portion of the test than their true positives on the recognition portion. Qualitatively, this may have to do with the participants finding the array of stimuli on the recognition portion of the test overwhelming or confusing. More quantitatively, earlier analyses had demonstrated that processing speed on a task involving sequential visual scanning was strongly predictive of RFIT scores (Fazio, Faris, Yamout, & McGovern, 2016), and, therefore, this may provide a more scientific explanation for the authors' qualitative observations.

Overall, findings indicate there is a relationship between age-related cognitive decline and performance on what is considered a fairly "easy" PVT. The relationship between diagnosis and RFIT failure strongly implicates declining memory abilities as a significant contributor, although some earlier exploratory analyses indicated that decline in processing speed may also be contributory (Fazio et al., 2016). Similarly, declines in attention or visual perception may likewise be underlying these age-related decrements in performance. Another possibility would be a decline in executive functioning, as one has to be able to identify the simple nature of the test in order to effectively implement it as a recall strategy. These relationships will need further exploration in future studies as if it can be determined why elderly individuals struggle with this test, then appropriate modifications could be made in order to improve its sensitivity and specificity with this population. For example, if processing speed is a significant issue, the stimulus card may need to be presented for a longer interval, or perhaps multiple trials would improve performance given the overall increase in exposure time.

The primary limitation of this study is the heterogeneous nature of the clinical data available. Although this represents a common scenario in clinical practice and therefore makes this data representative of a typical outpatient memory clinic, not all participants were administered the same tests in order to render a neuropsychological diagnosis, and they were diagnosed by two different clinicians, likely adding some variability to the diagnostic process. Similarly, the underlying etiologies of the MCI and MND diagnosed here were not neuropathologically confirmed and in many cases the evaluees had not had neuroimaging or other diagnostic tests (past routine lab work) at this point in the evaluative process. Referral bias may have also played some role in these results, as memory difficulties may be too easily viewed by some as a "normal" part of aging, leading to later referrals with higher likelihood of memory difficulties serious enough to result in a MCI/MND diagnosis.

A secondary limitation is that RDS is not the most robust measure to ensure the performance validity of this type of sample. A test such as the TOMM (Tombaugh, 1996) or the MSVT with GMIP (Green, 2004) may have had higher sensitivity. These tests, however, have the drawback of adding to evaluation time as they are freestanding measures, and this may produce some fatigue in older adults. Thus, these measures were not used in order to maximize the amount of useful clinical data collected given the clinical, rather than research, setting of these evaluations.

Looking ahead, it would be beneficial to have these findings independently replicated in order to verify their reliability prior to clinical use. Further exploration will also be needed in terms of their sensitivity to detect suboptimal effort, preferably both with a simulation group and a criterion group of older adults with motivation to feign cognitive deficits.

Taken together, this study represents a preliminary investigation into an area of burgeoning importance. More research needs to be conducted regarding the performance of older individuals on freestanding and embedded PVTs to determine the most psychometrically valid cut scores with this population. Until that time, conservative use and interpretation of these measures with elderly adults is warranted.

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Gov. Ex. 166

Brockman
10/2/21

A
B
C
1
2
3
← 3 punctuation

GOVERNMENT EXHIBIT
4:21-CR-009-GCH
No. 166

GOVERNMENT EXHIBIT
166

Brekman
10/2/21

Reyis

Brockman
10/2/21

A
B
C
1
2
3 ← 3 punctuation

Brockman
10/2/21

A
B
C
1
2
3 ← ? recommendation

Brockman 10/2/23

TP = 4
FP = 4

Gov. Ex. 167

Coin-in-the-Hand Test

Name: Brockman

Date: 10/2/21

Instructions: People often forget things easily when they are distracted, so this next test will measure how easily you can remember even after a distraction. I will put a coin in one hand [hold both hands out so the palms are facing up, then put a coin in one hand], and then I will close both my hands. I will then ask you to close your eyes and distract yourself by counting backwards from 10 to 1, counting aloud so I can hear you. After you reach 1, open your eyes, try to remember which hand the coin was in, and pointed at hand. The coin will always be in the same hand it was in before you closed your eyes. We will do this memory task 10 times even though it might be challenging at times. Are you ready for the first try?

[After each time the patient chooses a hand, open both hands and say either "that's right" or "that's wrong," depending on the patient's response.]

Right X
Left ✓
Left ✓
Right ✓
Right X
Left ✓
Right ✓
Left ✓
Right ✓
Left X

Total Errors: 3

he closed both hands, each time I did. Time-mimicing - ? echopraxia

GOVERNMENT EXHIBIT
4:21-CR-009-GCH
No. 167

GOVERNMENT EXHIBIT
167

Gov. Ex. 168

CONNERS CPT 3™

Continuous Performance Test 3rd Edition™

C. Keith Conners, Ph.D.

Assessment Report

Name/ID:	Robert Brockman2
Age:	80
Gender:	Male
Birth Date:	May 28, 1941
Administration Date:	October 2, 2021
Normative Option:	Gender Specific norms
Input Device:	Keyboard
Assessor's Name:	Guilmette
Medication/Notes:	

This Assessment Report is intended for use by qualified assessors only, and is not to be shown or presented to the respondent or any other unqualified individuals or used as the sole basis for clinical diagnosis or intervention. Administrators are cautioned against drawing unsupported interpretations. To obtain a comprehensive view of the individual, information from this report should be combined with information gathered from other psychometric measures, interviews, observations, and available records. This report is based on an algorithm that produces the most common interpretations of the obtained scores. Additional interpretive information is found in the *Conners CPT 3 Manual* (published by MHS).

Introduction

MHS

The Conners Continuous Performance Test 3rd Edition (Conners CPT 3™) assesses attention-related problems in individuals aged 8 years and older. During the 14-minute, 360-trial administration, respondents are required to respond when any letter appears, except the non-target letter "X." By indexing the respondent's performance in areas of inattentiveness, impulsivity, sustained attention, and vigilance, the Conners CPT 3 can be a useful adjunct to the process of diagnosing Attention-Deficit/Hyperactivity Disorder (ADHD), as well as other psychological and neurological conditions related to attention.

Validity of Administration

The Conners CPT 3 performs a validity check based on the number of hits and omission errors committed, as well as a self-diagnostic check of the accuracy of the timing of each administration. If there is an insufficient number of hits to compute scores, and/or if the omission error rate exceeds 25%, these issues will be noted. Also, the program will issue a warning message noting that the administration was invalid if a timing issue is detected.

The number of omission errors made by the respondent is highly unusual. Although such a high number of omissions may indicate a clinical impairment, there are other possibilities that may relate to the validity of the test. For example, the respondent may have been fatigued, misunderstood the instructions, or lacked the motivation to respond with full effort. Observations made of the respondent during the administration, as well as other data regarding the respondent, will help in assessing the validity of the administration. Also note that one or more scores could not be computed due to too few hits. The respondent may have disengaged from the task for a significant period of time during the administration. As a result, some interpretive text may be unavailable. **Readministration of the Conners CPT 3 is strongly recommended.**

Response Style Analysis

The variable *C* represents an individual's natural response style in tasks that involve a speed-accuracy trade-off. Based on his or her score on this variable, a respondent can be classified as having one of the following three response styles: a **conservative** style (T-score ≥ 60) of responding that emphasizes accuracy over speed; a **liberal** style (T-score ≤ 40) of responding that emphasizes speed over accuracy; or a **balanced** style (T-score = 41-59) of responding that is sensitive to both speed and accuracy. Based on Robert's responses, **he has a very conservative style of responding that emphasizes accuracy over speed (T-score = 90).** This response style is often associated with slower reaction times, more omission errors (failure to respond to targets), and fewer commission errors (incorrect responses to non-targets). The influence of Robert's very conservative response style on other Conners CPT 3 scores should be taken into consideration throughout the interpretation process.

T-score Guidelines

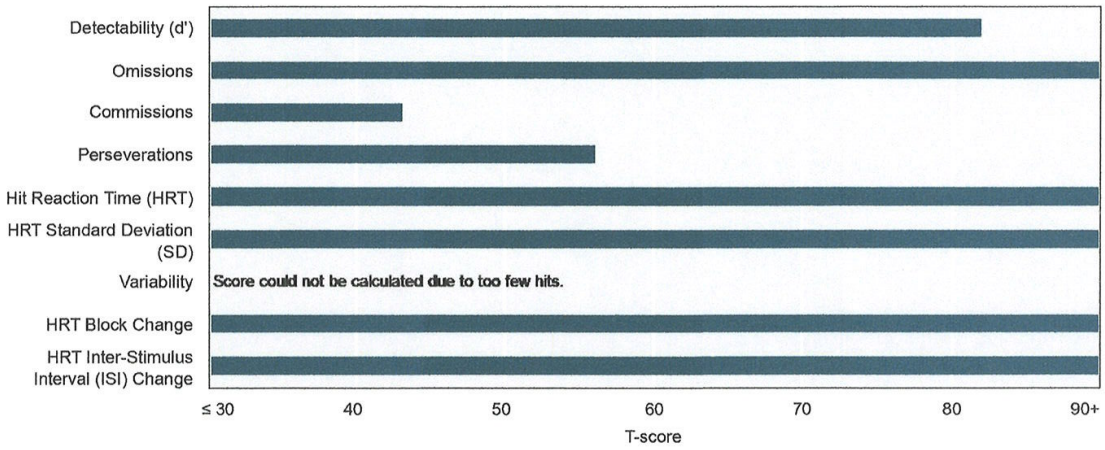
The guidelines in the following table apply to all T-scores in this report.

Guidelines			
T-score	For Hit Reaction Time (HRT)	T-score	For all other variables
70+	**Atypically Slow**	70+	**Very Elevated**
60-69	**Slow**	60-69	**Elevated**
55-59	A Little Slow	55-59	High Average
45-54	Average	45-54	Average
40-44	**A Little Fast**	< 45	Low
< 40	**Atypically Fast**		

Overview of Conners CPT 3 Scores

MHS

This section provides an overview of Robert's Conners CPT 3 scores.



Variable Type	Measure	T-score	Guideline	Interpretation
Detectability	d'	82	Very Elevated	**Pronounced difficulty** differentiating targets from non-targets.
Error Type	Omissions	90	Very Elevated	**Very high** rate of missed targets.
	Commissions	43	Low	**Good performance; below average** rate of incorrect responses to non-targets.
	Perseverations	56	High Average	**Slightly above average** rate of random, repetitive, or anticipatory responses.
Reaction Time Statistics	HRT	90	Atypically Slow	**Very slow** mean response speed.
	HRT SD	90	Very Elevated	**Very high inconsistency** in reaction times.
	Variability	?	?	?
	HRT Block Change	90	Very Elevated	**Very substantial** reduction in response speed in later blocks.
	HRT ISI Change	90	Very Elevated	**Very substantial** reduction in response speed at longer ISIs.

? = This score could not be calculated due to too few hits.

Summary: Relative to the normative sample, Robert was less able to differentiate targets from non-targets, made more omission errors, responded more slowly, displayed less consistency in response speed, displayed more of a reduction in response speed in later blocks and displayed more of a reduction in response speed at longer ISIs.

Overall, Robert has a total of 6 atypical T-scores, which is associated with a very high likelihood of having a disorder characterized by attention deficits, such as ADHD. Note that other psychological and/or neurological conditions with symptoms of impaired attention can also lead to atypical scores on the Conners CPT 3.

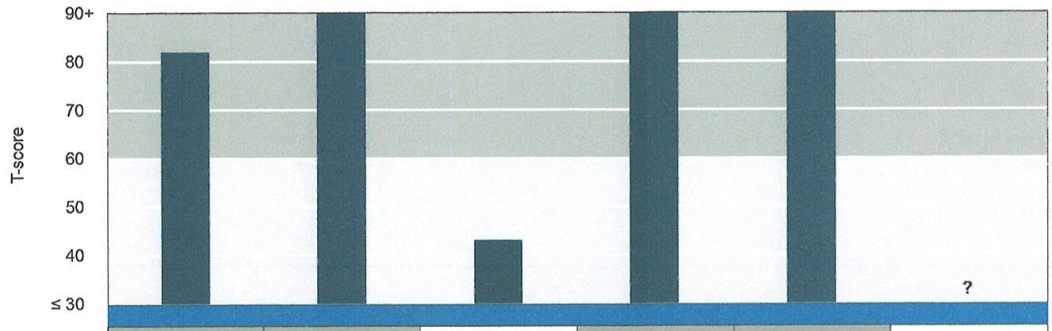
Robert's profile of scores and response pattern indicates that he may have issues related to:

- **Inattentiveness (Strong Indication)**
- **Sustained Attention (Strong Indication)**
- **Vigilance (Some Indication)**

Measures of Inattentiveness

MHS

This section summarizes Robert's scores on the inattentiveness measures and provides information about how he compares to the normative group. Indicators of inattentiveness on the Conners CPT 3 are poor Detectability (d'), a high percentage of Omissions and Commissions, a slow Hit Reaction Time (HRT), as well as high levels of inconsistency in response speed (Hit Reaction Time Standard Deviation [HRT SD] and Variability).



	Detectability (d')	Omissions	Commissions	HRT	HRT SD	Variability
T-score	82	90	43	90	90	?
Guideline	Very Elevated	Very Elevated	Low	Atypically Slow	Very Elevated	?

? = This score could not be calculated due to too few hits.

Detectability (d') measures the respondent's ability to differentiate non-targets (i.e., the letter X) from targets (i.e., all other letters). Robert's T-score is 82 and falls in the **Very Elevated** range. This result means that his ability to discriminate non-targets from targets was very poor when compared to the normative group. Poor ability to differentiate non-targets from targets is an indicator of inattentiveness.

Omissions result from a failure to respond to targets. Robert's T-score is 90 and falls in the **Very Elevated** range. This result means that he missed a much higher percentage of targets when compared to the normative group. Failure to respond to targets is an indicator of inattentiveness.

Commissions are made when responses are given to non-targets. Robert's T-score is 43 and falls in the **Low** range. This result means that he responded to a lower percentage of non-targets when compared to the normative group.

HRT is the mean response speed of correct responses for the whole administration. Robert's T-score is 90 and falls in the **Atypically Slow** range. This result means that his response speed was much slower than the normative group's response speed. This may indicate that Robert was not processing targets efficiently. Note that HRT may also be affected by response style; Robert's conservative response style may have contributed to the slower response speed. See the *Response Style Analysis* section of this report for more interpretive information.

HRT SD is a measure of response speed consistency during the entire administration. Robert's T-score is 90 and falls in the **Very Elevated** range. This result means that his response speed was much less consistent than the normative group. This suggests that Robert was more inattentive and processed stimuli less efficiently during some portions of the administration.

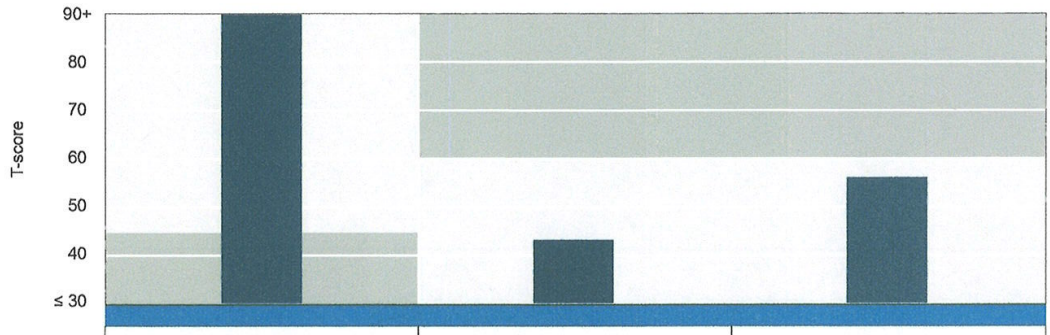
Variability, like HRT SD, is a measure of response speed consistency; however, Variability is a "within respondent" measure; that is, the amount of variability the individual shows in 18 separate segments of the administration in relation to his own overall HRT SD. This scale score could not be calculated due to too few hits.

Robert's scores on these measures strongly suggest that he may have problems with inattentiveness.

Measures of Impulsivity

MHS

This section summarizes Robert's scores on the impulsivity measures and provides information about how he compares to the normative group. Indicators of impulsivity on the Conners CPT 3 include a faster than normal Hit Reaction Time (HRT) in addition to a higher than average rate of Commissions and/or Perseverations.



	HRT	Commissions	Perseverations
T-score	90	43	56
Guideline	Atypically Slow	Low	High Average

HRT is the mean response speed of correct responses for the whole administration. Robert's T-score is 90 and falls in the **Atypically Slow** range. This result means that his response speed was much slower than the normative group's response speed. This may indicate that Robert was not processing targets efficiently. A slower than normal HRT is often related to inattentiveness rather than impulsivity. See the *Measures of Inattentiveness* section of this report for more interpretative information.

Commissions are made when responses are given to non-targets. Robert's T-score is 43 and falls in the **Low** range. This result means that he responded to a lower percentage of non-targets when compared to the normative group.

Perseverations are random or anticipatory responses. Robert's T-score is 56 and falls in the **High Average** range. This result means that he made slightly more perseverative errors when compared to the normative group.

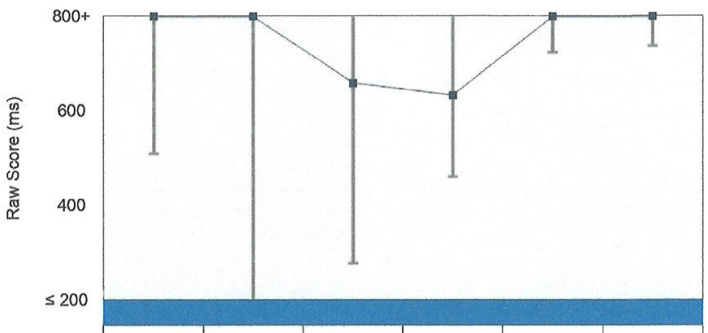
Robert's scores on these measures do not indicate a problem with impulsivity.

Measures of Sustained Attention

MHS

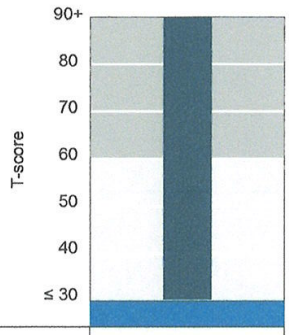
This section summarizes Robert's scores on the sustained attention measures. Sustained attention is defined as the respondent's ability to maintain attention as the administration progresses. A decrease in sustained attention across time is captured by atypical slowing in the respondent's Hit Reaction Times (HRT; as indicated by the variable HRT Block Change), as well as by increases in Omissions and Commissions in later blocks of the administration.

Hit Reaction Time by Block



	Block 1	Block 2	Block 3	Block 4	Block 5	Block 6
HRT (ms)	878	1025	659	633	1182	1204
HRT SD (ms)	369	949	384	173	458	466

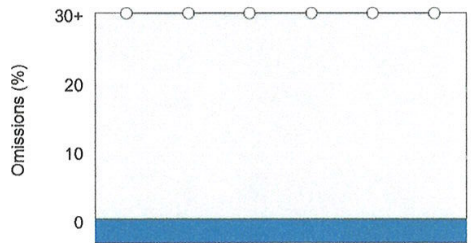
HRT Block Change



T-score	90
Guideline	Very Elevated

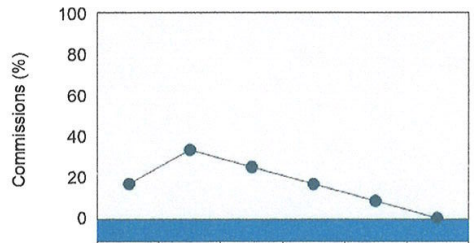
Note. ms = milliseconds; SD = Standard Deviation.

Omissions by Block



	Block 1		Block 2	Block 3	Block 4	Block 5	Block 6
Omissions (%)	56	<	81	77	81	75	79 *

Commissions by Block



	Block 1	Block 2	Block 3	Block 4	Block 5	Block 6
Commissions (%)	17	33	25	17	8	0

Note. The < symbol indicates that the error rate of the later block is significantly ($p < .10$) higher than the error rate of the previous block. The * symbol indicates that the error rate in Block 6 is significantly ($p < .10$) higher than the error rate in Block 1.

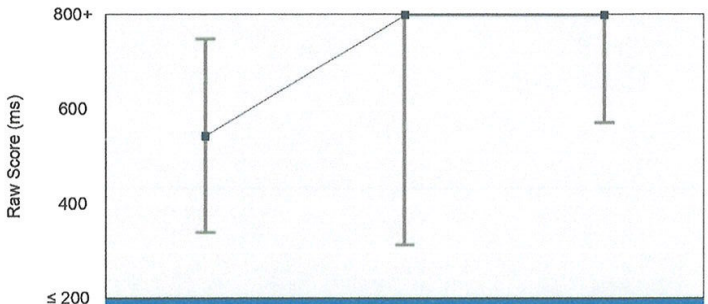
HRT Block Change indicates the change in mean response speed across blocks. Robert's T-score is 90 and falls in the **Very Elevated** range. This result means that he had a very substantial reduction in response speed in later blocks. In terms of error rates, Robert's omission errors increased significantly ($p < .10$) across blocks, but his commission errors did not signficantly increase across multiple adjacent blocks. **Robert's profile of scores on these measures indicates strong support for a problem with sustained attention.**

Measures of Vigilance

MHS

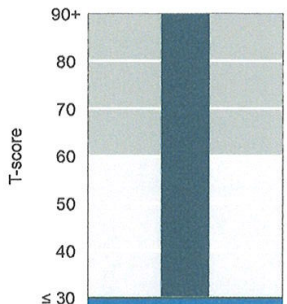
This section summarizes Robert's scores on the vigilance measures. Vigilance relates to the respondent's performance at varying levels of stimulus frequency (inter-stimulus intervals; ISIs), and is defined by the respondent's ability to maintain performance level even when the task rate is slow. This construct is captured by changes in the respondent's Hit Reaction Times (HRT), as indicated by the variable HRT ISI Change, as well as the observed pattern of Omissions and Commissions at various ISIs.

Hit Reaction Time by ISI



	1-second ISI	2-second ISI	4-second ISI
HRT (ms)	545	803	1122
HRT SD (ms)	206	489	550

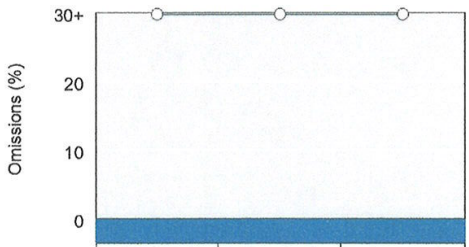
HRT ISI Change



T-score	90
Guideline	Very Elevated

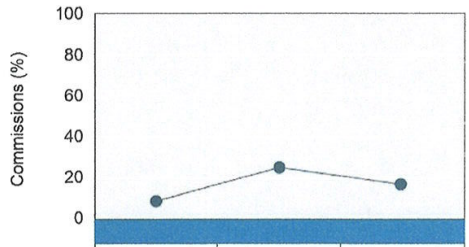
Note. ms = milliseconds; SD = Standard Deviation.

Omissions by ISI



	1-second ISI	2-second ISI	4-second ISI
Omissions (%)	82	86	56

Commissions by ISI



	1-second ISI	2-second ISI	4-second ISI
Commissions (%)	8	25	17

Note. No statistically significant differences were found in error rates between ISIs.

HRT ISI Change indicates the change in mean response speed at various ISIs. Robert's T-score is 90 and falls in the **Very Elevated** range. This result means that he had a very substantial reduction in response speeds at longer ISIs. There was no statistically significant increase in error rates across all three ISI levels. **Robert's profile of scores on these measures indicates some support for a problem with maintaining vigilance; that is, he had some problems with performance on trials with longer intervals between stimuli.**

Glossary

MHS

Response Style

C is a signal detection statistic that measures an individual's natural response style in tasks involving a speed-versus-accuracy trade-off. Based on his or her score on this variable, a respondent can be classified as having one of the following three response styles: a *conservative* style that emphasizes accuracy over speed; a *liberal* style that emphasizes speed over accuracy; or a *balanced* style that is biased neither to speed nor accuracy. Response style can affect scores such as Commissions and Hit Reaction Time (HRT), and should be taken into consideration during interpretation.

Detectability (*d'*)

d-prime (*d'*) is a measure of how well the respondent discriminates non-targets (i.e., the letter X) from targets (i.e., all other letters). This variable is also a signal detection statistic that measures the difference between the signal (targets) and noise (non-targets) distributions. In general, the greater the difference between the signal and noise distributions, the better the ability to distinguish non-targets and targets. On the Conners CPT 3, *d'* is reverse-scored so that higher raw score and *T*-score values indicate worse performance (i.e., poorer discrimination).

Omissions (%)

Omissions are missed targets. High omission error rates indicate that the respondent was not responding to the target stimuli due to a specific reason (e.g., difficulty focusing). Omission errors are generally an indicator of inattentiveness.

Commissions (%)

Commissions are incorrect responses to non-targets. Depending on the respondent's HRT, high commission error rates may indicate either inattentiveness or impulsivity. If high commission error rates are coupled with slow reaction times, then the respondent was likely inattentive to the stimulus type being presented and thus responded to a high rate of non-targets. If high commission error rates are combined with fast reaction times, the respondent was likely rushing to respond and failed to control his or her impulses when responding to the non-targets. In the latter case, high commission error rates would reflect impulsivity rather than inattentiveness.

Perseverations (%)

Perseverations are responses that are made in less than 100 milliseconds following the presentation of a stimulus. Normal expectations of physiological ability to respond make it virtually impossible for a respondent to perceive and react to a stimulus so quickly. Perseverations are usually either slow responses to a preceding stimulus, a random response, an anticipatory response, or a repeated response without consideration of the task requirements. Perseverations may be related to impulsivity or an extremely liberal response style. Perseverations are, therefore, likely the result of anticipatory, repetitive, or impulsive responding.

Hit Reaction Time (HRT)

HRT is the mean response speed, measured in milliseconds, for all non-perseverative responses made during the entire administration. An atypically slow HRT may indicate inattentiveness (especially when error rates are high), but it may also be the results of a very conservative response style. Alternatively, a very fast HRT, when combined with high commission error rates, may indicate impulsivity.

Hit Reaction Time Standard Deviation (HRT SD)

HRT SD measures the consistency of response speed to targets for the entire administration. A high HRT SD indicates greater inconsistency in response speed. Response speed inconsistency is sometimes indicative of inattentiveness, suggesting that the respondent was less engaged and processed stimuli less efficiently during some parts of the administration.

Variability

Variability, like HRT SD, is a measure of response speed consistency; however, Variability is a "within respondent" measure (i.e., the amount of variability the respondent showed in 18 separate sub-blocks of the administration in relation to his or her overall HRT SD score). Although Variability is a different measure than HRT SD, the two measures typically produce comparable results and are both related to inattentiveness. High response speed variability indicates that the respondent's attention and processing efficiency varied throughout the administration.

Hit Reaction Time Block Change (HRT Block Change)

HRT Block Change is the slope of change in HRT across the six blocks of the administration. A positive slope indicates decelerating reaction times as the administration progressed, while a negative slope indicates accelerating reaction times. If reaction times slow down, as indicated by a higher HRT Block Change score, the respondent's information processing efficiency declines, and a loss of sustained attention is indicated.

Omissions by Block

Omissions by Block (raw score only) is the rate of the respondent's missed targets in each of the six blocks. An increase in omission error rate in later blocks indicates a loss of sustained attention.

Commissions by Block

Commissions by Block (raw score only) is the rate of the respondent's incorrect responses to non-targets in each of the six blocks. An increase in commission error rate in later blocks indicates a loss of sustained attention.

Hit Reaction Time Inter-Stimulus Intervals Change (HRT ISI Change)

HRT ISI Change is the slope of change in reaction time across the three ISIs (1, 2, and 4 seconds). A positive slope indicates decelerating HRT at longer intervals; whereas, a negative slope indicates accelerating HRT at longer intervals. A higher HRT ISI Change score means that the respondent's information processing efficiency declined with longer pauses between stimuli, and a loss of vigilance is indicated. A significant change in response speed at the different ISIs may indicate that the respondent was having trouble adjusting to changing task demands. Sometimes, this finding relates to activation/arousal needs; some respondents may be more efficient in a busier/more stimulating environment (e.g., during the 1-second ISI) than in a less active environment where the stimuli are presented less frequently (e.g., during the 4-second ISI), or vice-versa.

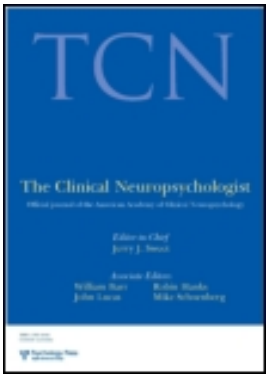
Omissions by ISI

Omissions by ISI (raw score only) is the rate of missed targets in each of the three ISI trial types. An increase in omission error rate on trials with longer ISIs indicates a loss of vigilance.

Commissions by ISI

Commissions by ISI (raw score only) is the rate of incorrect responses to non-targets in each of the three ISI trial types. An increase in commission error rates on trials with longer ISI indicates a loss of vigilance.

Gov. Ex. 169



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American Academy of Clinical Neuropsychology Consensus Conference Statement on the Neuropsychological Assessment of Effort, Response Bias, and Malingering

Robert L. Heilbronner, Jerry J. Sweet, Joel E. Morgan, Glenn J. Larrabee, Scott R. Millis & Conference Participants

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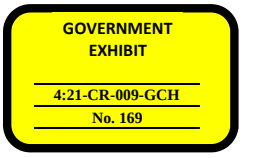

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AMERICAN ACADEMY OF CLINICAL NEUROPSYCHOLOGY CONSENSUS CONFERENCE STATEMENT ON THE NEUROPSYCHOLOGICAL ASSESSMENT OF EFFORT, RESPONSE BIAS, AND MALINGERING

Robert L. Heilbronner, Jerry J. Sweet, Joel E. Morgan, Glenn J. Larrabee, Scott R. Millis, and Conference Participants[1]

During the past two decades clinical and research efforts have led to increasingly sophisticated and effective methods and instruments designed to detect exaggeration or fabrication of neuropsychological dysfunction, as well as somatic and psychological symptom complaints. A vast literature based on relevant research has emerged and substantial portions of professional meetings attended by clinical neuropsychologists have addressed topics related to malingering (Sweet, King, Malina, Bergman, & Simmons, 2002). Yet, despite these extensive activities, understanding the need for methods of detecting problematic effort and response bias and addressing the presence or absence of malingering has proven challenging for practitioners. A consensus conference, comprised of national and international experts in clinical neuropsychology, was held at the 2008 Annual Meeting of the American Academy of Clinical Neuropsychology (AACN) for the purposes of refinement of critical issues in this area. This consensus statement documents the current state of knowledge and recommendations of expert clinical neuropsychologists and is intended to assist clinicians and researchers with regard to the neuropsychological assessment of effort, response bias, and malingering.

[1]Conference Participants (by working group): *Definitions and Differential Diagnosis*: Robert L. Heilbronner (Chair), Kevin J. Bianchini, Paul Kaufmann, Daniel J. Slick, H. Gerry Taylor. *Ability Issues*: Jerry J. Sweet (Chair), Kyle Brauer Boone, Shane S. Bush, Kevin W. Greve, Thomas J. Guilmette. *Somatic Issues*: Glenn J. Larrabee (Chair), Manfred F. Greiffenstein, Nathaniel W. Nelson, Julie Suhr, David T. R. Berry*. *Psychological Issues*: Joel E. Morgan (Chair), Robert L. Denney, Robert J. McCaffrey, Christopher L. Grote, Roger O. Gervais. *Research Evidence and Scientific Issues*: Scott R. Millis (Chair), William B. Barr, Jacobus Donders, Grant L. Iverson, Martin L. Rohling. *External Review Panel*: Laurence M. Binder, Paul Lees-Haley, George J. Demakis, Wiley Mittenberg, Richard I. Frederick.

*Dr. Berry was unable to attend the meeting, but reviewed and approved the draft work of the working group.

Conference participants were not compensated for their participation or reimbursed for expenses incurred as a result of participation. The authors acknowledge the assistance of Dawn Giuffre Meyer in the preparation of this manuscript.

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DEVELOPMENT OF THE CONSENSUS CONFERENCE AND THE CONSENSUS STATEMENT

Organizers Robert Heilbronner and Jerry Sweet developed the idea for the Consensus Conference, with the need for the conference based on the degree of interest expressed on the part of forensic neuropsychology experts, clinical researchers, and both junior and senior colleagues in the field. It was apparent from high attendance at relevant continuing education presentations during the past decade that a substantial proportion of practicing neuropsychologists were regularly seeking guidance based on relevant research and the advice of identified expert colleagues. The desired guidance appeared to focus primarily on the detection of problematic effort and other forms of response bias, such as biased symptom reporting, related to diagnosing or ruling out neuropsychological malingering. The American Academy of Clinical Neuropsychology (AACN) Board of Directors approved sponsorship of the proposed conference in June 2007, entrusting Heilbronner and Sweet with the organization of the conference, to be held at the annual meeting of the Academy in Boston in June 2008, with the resulting statement to be published in the official journal of the Academy, *The Clinical Neuropsychologist*.

The primary goal of the consensus conference was to produce a consensus statement, developed by prominent expert neuropsychologists, based on sound practice principles and reflecting the broad scientific literature relevant to the measurement of effort, response bias, and neuropsychological malingering. There is a common misperception that clinical neuropsychologists often find points of disagreement on clinical and forensic topics. In actuality all science-driven healthcare specialties create progress by a process of challenging current and new ideas through intellectual discourse and empirical hypothesis testing. In this manner, clinical neuropsychology is no different. For this reason it was expected that clinical and forensic experts who are involved regularly in presenting at professional meetings and/or who frequently publish empirical peer-reviewed research on the subject matter of the conference would in fact be able to arrive at points of consensus. In turn, these points of consensus can aid neuropsychology practitioners and researchers.

The organizers identified five relevant content areas to be addressed at the consensus conference and reviewed a lengthy bibliography (Sweet, 2009) to identify broad and specific topic experts for each. Relevant to each content area, literature citations and professional conference programs were reviewed to identify relevant experts. Of the eventual list of 25 experts identified, five individuals (Heilbronner, Larrabee, Millis, Morgan, Sweet) were identified as content section working group chairs. These chairs met in February 2008 to discuss the proposed structure and process of the conference. During this initial meeting, a number of consensus statements published previously by other professional organizations were reviewed.

The working group chairs agreed in advance to identify, with the assistance of the members of their working groups, pre-conference reading materials. The list of each group's pre-conference readings (together with the agenda of the conference) was circulated to all 25 invitees before the meeting, and can be found in the Appendix.

PROCESS OF CREATING CONSENSUS AND WRITING THE CONSENSUS STATEMENT

The participants of the conference met on June 17, 2008, the day prior to the AACN Annual Conference and Workshops in Boston. The conference began with a review of the general approach and purpose of a consensus conference.

Conflict of interest forms were completed at the beginning of the conference by all participants because of the possibility that specific commercially available tests authored by and accruing royalties to one or more participants might be mentioned in the eventual consensus statement. Comparison of potential conflicts of interest with the final draft of the statement did not reveal conflict of interest for any participant. More generally, the salience and relevance of active involvement in forensic activities was a qualification for invitation to be part of the consensus conference, either as a participant or as an external reviewer. The very nature of a consensus conference, which requires agreement among all contributors, effectively negates the influence of any single participant's or external reviewer's personal preference or bias in forensic practice.

The conference alternated between working group breakouts and overall group discussions. Each working group had been provided an outline in advance, to assist in the completion of a work product for each group. From this outline, each group began to develop its portion of the consensus statement. At multiple points in the initial full day of the conference, working group chairs summarized points of consensus to the entire group of participants to assess the larger group's viewpoints and possible consensus. After the end of the first day, working group chairs constructed a PowerPoint presentation, which was presented to a large audience of general AACN conference attendees in the form of a 3-hour workshop on June 19. Each of the working group chairs presented points of consensus relevant to their content areas, for discussion with and input from the audience members. The conference participants reconvened on the afternoon of June 20, following the close of the AACN meeting, to discuss and integrate the input from the workshop attendees. Subsequently, working groups worked via phone and e-mail to construct the five sections of this statement, which the organizers integrated into one document. The draft statement then underwent additional review by all the conference participants, as well as a five-member independent panel of external reviewers.[2] Thus, the content of this Consensus Conference Statement reflects the input of 30 clinical neuropsychologists with expertise relevant to the neuropsychological assessment of effort, response bias, and malingering.

DEFINITIONS AND DIFFERENTIAL DIAGNOSIS

As noted in the introduction to this Consensus Conference Statement, the majority of research and clinical work on the neuropsychological assessment of effort, response bias, and malingering has occurred during the last 20 years. During this time various descriptors and terms have been applied to convey salient relevant

[2] External Review Panel: Laurence M. Binder, Paul Lees-Haley, George J. Demakis, Wiley Mittenberg, Richard I. Frederick.

concepts, sometimes in a manner that is helpful, and other times less so. We aspire in this section to clarify terminology, in a manner that facilitates an understanding of relevant concepts for clinical practice and scientific investigation.

In the overall process of conducting an assessment, when there is concern about insufficient effort or malingering, a distinction can be made between the processes of ''detection'' and ''diagnosis.''

Detection

Neuropsychologists are concerned with cognitive, emotional, and physical capacities and symptoms. When neuropsychologists use indicators developed in studies that feature malingering and non-malingering groups, the classification accuracy statistics describe the capacity of the indicator (at different score levels) to detect malingering (see more about this below in the ''Research Design'' section). The *detection* of malingering can occur in the neuropsychological assessment of any individual during the course of assessing symptoms, abilities/capacities, and functionality. There are two strategies employed by examinees that are of interest, which undermine the validity of the examination. These involve attempts to intentionally create the appearance of disability, which is a common primary goal of malingering (Bianchini, Greve, & Glynn, 2005). These strategies are to intentionally diminish or reduce capability and intentionally exaggerate symptom complaints.

Various terms have been used by researchers and clinicians to describe the behaviors of interest in the identification of intentionally exaggerated symptoms and diminished or reduced capability. In particular, when related to the validity of ability measurements, clinical researchers have chosen a number of words to convey problems with *effort*. These terms include, among others: insufficient effort, inadequate effort, and poor effort. There does not appear to be consensus on the preferred descriptor for effort, yet it can be noted in these three examples that a useful descriptor of problematic effort is one that clearly conveys a substantial negative impact that has the potential of invalidating measurement of ability. Measures used to identify problematic effort are often identified as *effort tests*, which are considered to be in a category of measures that evaluate validity of symptoms, known as *symptom validity tests* (SVTs). In this vein, it is important to clarify that these *effort tests* actually require little effort or ability, as they typically are normally performed (and in some cases, perfectly performed) by a wide range of patients who have bona fide neurologic, psychiatric, or developmental problems. Failure on such measures indicates that not enough effort was expended in the direction of capable performance. Some experts prefer to consider that failure on an effort test shows that considerable effort to perform poorly was expended. Validity indicators that are ''built in'' to standard neuropsychological measures are referred to as *embedded measures*. The focus of specialized symptom validity tests and embedded indicators is considered to be the measurement of *performance validity* or *response validity*. Most procedures designed to detect a response bias relevant to malingering are aimed at identifying a *negative response bias*. This element of the detection process dealing with invalid performances on measures of neuropsychological function will be discussed more extensively in the Ability Issues section of this Consensus Conference document.

Intentional exaggeration of symptom complaints is a separate issue in the malingering detection process. Common terms related to this examinee behavior include *symptom exaggeration* and *symptom magnification*. Indicators of symptom exaggeration are often incorporated into psychological tests that elicit self-report of symptoms. This topic will be discussed more extensively in the Somatic and Psychological Issues sections of this document.

When considering neuropsychological test performance, concerns regarding effort are frequently related to consideration of whether an examinee is malingering. However, simply equating "poor effort" with malingering is an oversimplification. For example, an examinee who is malingering may simply put forth poor effort on neuropsychological testing as a component of his or her approach to malingering. A different examinee, also malingering, may expend considerable effort to avoid detection while being examined. This is a complex conceptual issue. The process of detecting malingering is one in which consideration is given to multiple dimensions of behavior that differentiate malingering from other entities, such as factitious disorder, conversion disorder, cogniform disorder (Delis & Wetter, 2007), and somatoform disorder.

The dimension of *effort level* can be conceptualized as occurring on a continuum and can vary within and across tests and also across other types of behaviors observed during an assessment (e.g., level of effort expended to recall autobiographical information during the interview or level of effort expended in an attempt to convincingly present with episodic memory deficits during formal testing). Other related concepts (e.g., volitional vs non-volitional, internal goal vs external goal, etc.) are best considered not as present or absent, but rather as being continua denoting the relative influence of opposite factors. For example, in some instances, examinee behavior (e.g., intentionally feigning deficits) may be for the purpose of meeting internal psychological needs (e.g., factitious disorder) or toward obtaining an external, material reward (e.g., malingering). In other instances, either an internal or external goal may be pre-eminent, or both may be equally important. Note that these dimensions can be thought of as underlying very specific behaviors, such as an examinee's response to a given test item or specific interview question. These dimensions can also be used to characterize an examinee's behavior more broadly, such as during performance on a given test or test battery or during the course of an interview.

Diagnosis

In considering the diagnosis of malingering, the clinician is explicitly making a determination of intent: more specifically, a determination of intentionally exaggerated symptoms and/or intentionally diminished capability with the goal of obtaining an external reward. The committee reached a consensus that, through application of relevant psychological and neuropsychological science, clinicians *can diagnose* malingering in some examinees. Clinicians using this scientific foundation/body of knowledge can differentiate intentionally exaggerated presentations/disorders/conditions/diagnoses (e.g., malingering and factitious disorder) from unintentionally exaggerated presentations (e.g., somatoform pain disorder, cogniform disorder). To do this, the context of the evaluation and overall presentation of

the examinees, including background information, history information gathered during interview, observations, neuropsychological tests, and measures of response bias, should be considered in this process.

For the diagnosis of malingering, there are published diagnostic classification systems (e.g., Bianchini et al., 2005; Slick, Sherman, & Iverson, 1999) that better represent current neuropsychological knowledge regarding malingering indicators than the current version of the American Psychiatric Association Diagnostic and Statistical Manual, Fourth Edition-Text Revision (DSM-IV-TR; APA, 2000). Although likely to evolve and undergo refinement across time, such neuropsychological diagnostic systems offer a reliable means of operationalizing diagnostic decisions related to the determination of malingering and are consistent with appropriate clinical (*AACN Practice Guidelines for Neuropsychological Assessment and Consultation*; AACN, 2007) and forensic (*Specialty Guidelines for Forensic Psychologists*; Committee on Ethical Guidelines for Forensic Psychologists, 1991) guidelines in explicitly recommending that clinicians incorporate multiple sources of data and information. Empirically based systems are recommended when making a diagnosis, as they provide increased reliability of the classification accuracy of findings of the various validity indicators. It is recommended that clinicians be familiar with the psychological and neuropsychological literature related to the classification accuracy of validity indicators and how well the sample of a given study generalizes to the individual being examined. Within this Consensus Conference Statement, the term *diagnosis* is used in a manner consistent with use in the relevant neuropsychological literature (e.g., Bianchini et al., 2005; Slick et al., 1999). In the current version of the DSM-IV-TR (APA, 2000), malingering is assigned a *V-code* indicating that it is not a psychiatric illness and that a disease process is not implied with the designation. Although we use the term *diagnosis,* we agree with the DSM-IV-TR that malingering is *not* a mental illness or disorder. In this sense, our use of the term diagnosis refers to assigning malingering as a designation and a descriptive term to describe intentional exaggeration, and is not a description of a disease process.

Because the diagnosis of malingering involves an explicit consideration of the *purpose* of a given behavior, the committee recognizes that an important part of the diagnostic process involves a consideration of the *context* of the evaluation. In a routine clinical context the primary gain of a *patient* is relief of some form of physical or emotional symptom. Some terms have been used and misused in describing important aspects of the forensic context, including: *gain, secondary gain*, *external gain*, and *financial gain*. In order to reach a determination that intentional exaggeration is produced as a manifestation of malingering (versus factitious disorder) the clinician must determine that the patient has something to gain from being impaired. This potential is sometimes referred to as *secondary gain*, which is often mistakenly used as a synonym for malingering. Secondary gain differs from primary, typically emotional, gain, which is often the characteristic motivation for the intentional exaggeration in factitious disorders. The concept of secondary gain is well described in the DSM-IV-TR (APA, 2000) as including: financial reward, compensated time away from work, avoidance of military duty, relief from legal consequences, and obtaining medications/narcotics. It is usually not difficult to determine whether the patient has the capacity to gain from his or her symptoms or

claimed impairment. Typical contexts that involve secondary gain potential include litigation, disability claims, criminal prosecution, and worker's compensation claims. Military service and post-service involvement with the Department of Veterans Affairs can also involve secondary gain potential, as noted in a relevant task force report that includes mention of the appropriateness of evaluating symptom validity and response bias (McCrea et al., 2008). The committee recommends that the use of the term secondary gain be limited to a *description of the context within which the evaluation is taking place* and not used as a synonym for malingering.

The term *malingering* is descriptive. In some instances malingering can be adaptive. As previously discussed, the differential diagnosis of malingering must take into consideration the role of intent. The best way to assess intent is by ruling out other possible conditions (e.g., psychological, neurological, developmental) that might otherwise explain the suspicious behavioral presentation and by requiring the presence of multiple improbable performances and/or atypical symptomatic complaints. The differential diagnosis of malingering is a clinical process that: (1) requires careful analysis on the part of the examiner, (2) is based on objective criteria; (3) incorporates indicators that have established classification accuracy, and (4) combines clinical judgment with the results of scientifically validated measures in this process.

An attempt to generalize one set of behaviors (e.g., malingering behaviors) to a specific case involves making an inference. As is true for clinical decision making, when making a determination of malingering, this inference, and the data set on which it is based, including formal test results, must have a strong scientific foundation. A salient judgment concerns how well the published literature generalizes to the examinee. Staying abreast of current knowledge facilitates scientifically based inferences about the behavior of examinees, both within and outside of the test environment.

There is consensus regarding the existence of a research foundation that neuropsychologists can rely on in determining an examinee's intent to exaggerate symptoms or perform below their capabilities in testing. Scientific literature in the field of clinical neuropsychology indicates that neuropsychologists possess evidence-based methods for detecting intentional exaggeration of cognitive and emotional functioning and somatic complaints that can be used in an empirical, clinical decision-making process. Relying on this scientific body of knowledge, neuropsychologists are capable and qualified to diagnose malingering.

The determination that malingering is present often involves the application of scientific results to a forensic question. This information can be used to assist the trier-of-fact (e.g., judge, jury) in a legal decision-making process. Neuropsychologists remain mindful of the important difference between scientifically based clinical decisions and legal adjudication. Moreover, they recognize and respect the laws and customs of the jurisdiction in which they practice when describing the behavioral presentation at issue. Finally, in performing a competent differential diagnosis of malingering, the process is best served by a dispassionate consideration of all factors related to the context in which the behavior occurs. This includes consideration of the diversity of examinees, in terms of language, culture, education, and ethnic minority

status, which may not be matched by the diversity of neuropsychological examiners in these same variables (Romero et al., 2009).

ABILITY ISSUES

Conceptual and operational definitions

Misrepresentation of abilities in any neuropsychological domain of ability (memory, sensorimotor, language, etc.) through performance, or self-report regarding performance capabilities, represents response bias. Whereas many of the ability domains could be termed *cognitive*, some, such as motor function, are more accurately depicted as *abilities*. When in the presence of the potential for external gain the valence of the response bias is negative (i.e., in the direction that would increase the likelihood or magnitude of external gain), malingering needs to be considered. External gain can take the form of monetary reward, non-monetary incentive (e.g., drug seeking), avoidance of responsibility, or escape from undesirable or intolerable conditions.

Negative response bias involving ability performance is operationalized by failure to surpass the thresholds of effort tests or embedded validity indicators within ability tests and/or response bias validity scales within self-report measures. Alternatively, gross disparity between ability test performance and real-world activities could represent response bias. With regard to ability performances, these invalid presentations: (1) are not fully explained by brain dysfunction, (2) are not reasonably attributable to variables that may in some instances moderate (e.g., education, age) or may in some instances confound (e.g., fatigue, psychological conditions) performances on ability tests, and (3) are significantly worse than, or at least different in degree or pattern from, performance standards known to reflect genuine neurological disorder.

Review of records, clinical interview, and comparison of test results to "real-world" behavior can be essential in addressing the possibility of malingering. Related to interviewing, the concepts of *false history and incomplete history* are important. These represent more than normal errors of omission in providing history or inexact provision of history. Providing a false history involves misrepresenting relevant information that is salient to issues central to a forensic consultation. Providing incomplete history involves omitting important information that could prove central to consideration of the causes of, or alternative explanations for, claimed symptoms and alleged disability. In the process of evaluating an examinee's credibility as a historian, it is important to assess whether the individual is providing a reliable and valid account of his/her history.

Tests and other psychometric procedures relied on by clinicians in judging response validity must themselves have proven validity. In fields of scientific investigation, such as clinical neuropsychology and related areas, there are multiple peer review journals in which the validity of tests and procedures related to neuropsychological response bias have been and continue to be the subject of empirical scrutiny (Sweet et al., 2002). The size of the neuropsychological literature related to assessment of response bias in the measurement of abilities and the overall quality of this literature is substantial and well developed; this area

of investigation should not be viewed as "experimental" or nascent. Indeed, the relevant scientific literature is broad and varied in its depiction of the effectiveness of specific measures, with a range of classification accuracy results. Clinicians need to be aware of these studies well beyond the original research contained within test manuals or initial publications of applications, and this requires staying current with the scientific literature in this area. Clinicians need to assign weight to specific results according to the rigor of the studies and the relevance of samples studied to the clinician's case at hand. It is expected that the quality and amount of existing research will guide the clinician's choice of measures for use in individual cases, with greater weight being given to indicators that have proven validity across multiple studies.

Types of assessment methods related to evaluating response validity of abilities

Methods of evaluating *performance*

Stand-alone cognitive effort tests. These measures have been developed specifically to evaluate task performance validity. Additional testing time is required for this class of tests. There is now abundant research evidence that stand-alone cognitive effort tests are extremely useful within forensic evaluations, which have been shown to be associated with a high risk of invalid responding. Therefore, the additional testing time is warranted, and "medically necessary" (Bush et al., 2005).

Forced-choice stand-alone cognitive effort measures are those that limit the examinee to choosing one of a fixed number of responses. With regard to stand-alone cognitive effort testing, most measures offer two response choices, such that a chance performance can be determined. Numerous clinical researchers (e.g., Greve, Binder, & Bianchini, 2009; Mittenberg, Patton, Canyock, & Condit, 2002; Slick, Tan, Strauss, & Hultsch, 2004) have investigated the base rates on forced-choice measures of *significantly below* chance performance (see statistical discussion by Frederick & Speed, 2007), which has been viewed as evidence of deliberate under-performance, and when occurring within a secondary gain context, supports a conclusion of malingering. As shown in additional research (e.g., Thompson, 2002; Tombaugh, 2002), it is now well known that invalid performance can be identified using thresholds that are well above a level that is significantly below chance.

Non-forced-choice stand-alone cognitive effort measures are those that allow a range of responses and may evaluate random responding, unrealistically slow or erroneous responding, and inconsistency of response patterns when compared to performances from well-documented disorders evaluated in a non-forensic context.

Embedded indicators within ability tests. This category of measures refers to validity indicators derived from standard clinical ability tests that have shown value in identifying non-credible or disingenuous performances. There are two forms: indicators developed for this specific purpose and traditional standard scores that have been found in post-release research to be sensitive to insufficient effort. To date, the majority of embedded indicators have been developed in

post-release research. The formats of these embedded measures are broad and can include consideration of forced-choice responding.

There are too many effort measures and embedded validity indicators with proven validity to list within this statement; extensive lists are available elsewhere (cf. Sweet, 2009). Effort measures have been the subject of numerous empirical studies, which have been summarized in reviews and textbooks (e.g., Boone, 2007; Hom & Denney, 2002; Larrabee, 2007) and meta-analytic research (Vickery, Berry, Inman, Harris, & Orey, 2001). Similarly, numerous embedded validity indicators have been the subject of significant research and related reviews (e.g., Babikian, Boone, Lu, & Arnold, 2006; Heinly, Greve, Bianchini, Love, & Brennan, 2005; Iverson & Tulsky, 2003).

Methods of evaluating *self-report*. Individuals with and without brain disorders may complain of diminished abilities. For example, it is common for depressed individuals to complain of decreased concentration and memory abilities (e.g., Otto et al., 1994). Conversely, patients with severe TBI may minimize their deficits due to limited awareness. No examiner in any discipline is required to simply accept self-reported facts and history of examinees. The validity of *self-reported* disability and symptoms needs to be evaluated, especially when such complaints occur in a forensic context. When evaluating the validity of self-report, if possible, clinicians include measures that possess an internal means of assessing response bias.

Disorder-specific inventories. Interest in specific conditions that can become chronic, such as pain and post-traumatic stress disorder (PTSD), has generated a small but growing number of narrowly designed inventories that are intended to compare an individual's responses to those of patients who have been given the same diagnosis (e.g., PTSD) or who experience the same type of symptom (e.g., pain). Some of these inventories solicit information pertaining to abilities. In order to be considered appropriate for use, it is recommended that these instruments have validity scales that produce acceptable classification statistics, including sensitivity, specificity, positive predictive power, and negative predictive power. In general, disorder-specific inventories and symptom checklists that do not contain effective means for determining response bias and possible response invalidity should not be used in isolation. If, during an examination, self-report measures containing effective validity scales show clear and consistent invalid responding, data from self-report instruments that have no validity scales should not be relied on.

General personality inventories. The fields of clinical psychology and clinical neuropsychology have a long history of using general personality inventories that have a strong research foundation and well-established validity scales. These inventories are not equivalent in application to all conditions, requiring that clinicians keep abreast of relevant research. In the presence of signs of obvious response bias, whether negative or positive, clinicians should be appropriately conservative in interpreting clinical scales from these inventories. In the presence of response bias that is strong enough to invalidate responses on clinical scales, the clinical results of the inventory should not be interpreted.

Effort and malingering as applied to assessment of abilities

There is consensus regarding the existence of malingering. There is also consensus that the term malingering is appropriate for use in some instances. There is consensus regarding the meaning of significantly below-chance findings and what has been referred to as a "compelling inconsistency" (Bianchini et al., 2005), and both are viewed as individually reflecting a deliberate attempt to misrepresent one's abilities for which there are no alternative explanations. Intent may also be inferred as a result of the combined improbability of events, rather than a single definitive indication of intent (Larrabee, Greiffenstein, Greve, & Bianchini, 2007). Evidence that may be considered in the process of a differential diagnosis involving the possibility of malingering of abilities includes gross (1) disparity between real-world observations and either test performance or self-report, (2) inconsistency between type or severity of injury and test performances, (3) inconsistency between an individual's behavior when he/she is aware of being evaluated versus when not aware of not being evaluated, and (4) inconsistency across serial testings that cannot be explained by an underlying neurological process or known psychiatric condition. Whether a clinician is comfortable using the term "malingering" or not, the decision as to whether a case reflects malingering should have a basis in scientific research and related peer-reviewed assessment approaches.

The importance of evaluating performance validity when assessing abilities

Because psychometric indicators of performance invalidity are typically set to reduce false positive outcomes, positive findings (i.e., when credible thresholds have been surpassed) are more meaningful than negative findings. That is, a positive finding tends to "rule in" insufficient effort, whereas a negative finding may or may not "rule out" insufficient effort. However, not all instances of insufficient effort are indicative of malingering (Slick et al., 1999; Sweet, 1999). For example, a single indication of insufficient effort within a large battery of tests could indicate a transient effort problem that might not lead to the general conclusion that malingering is present, which is a decision based more broadly on all available information.

Sufficient effort is needed on every ability test in order to produce valid results. Moreover, effort is not a static concept, but rather a dynamic phenomenon that may vary throughout the examination. As such, *ideally*, effort should be evaluated repeatedly, if not continuously, throughout the course of an examination (Boone, 2009). When inconsistent or variable effort is shown to be present at any point during an evaluation, a reasonable and conservative conclusion is that all performances and obtained test scores may underestimate actual abilities, even those that occurred during apparent periods of adequate effort.

When the term malingering is applied to ability performances or self-report of abilities, it is not expected that malingering automatically or necessarily explains all of the individual's behaviors and presentations that are being scrutinized. For example, even though an examinee is found not to be credible on ability

testing, findings of depression on personality inventories may still be accurate. However, these other data may need to be viewed with caution and corroborated by additional outside sources of information. Moreover, performances on various neuropsychological tests used in a particular examination in which non-credible performances are present are best viewed as a lower bound estimate of actual level of ability (cf. Bush et al., 2005). Last, when the term malingering is applied, its use does not presume to explain all previous or subsequent behavior. The determination of the degree to which malingering is wholly, or only partly, explanatory is specific to the current evaluation.

Clinicians should be well apprised of the substantial relevant literature and be able to judge when it is appropriate to conclude that evidence of malingering is present. Whether a conclusion of malingering is made or not, when there is evidence of negative response bias there is consensus that determinations such as *non-credible*, *invalid*, and *implausible* can be made reliably on the basis of test results when they are viewed in a clinical context. When present, such invalid performances preclude the use of those data as a basis for: (1) opinions with regard to attribution to the cause at issue (e.g., accident, injury), (2) the nature and extent of possible deficits and disability, and (3) guiding treatment or evaluating treatment effectiveness.

Whether the context of a neuropsychological evaluation is clinical or forensic, the validity of self-report of abilities is an important source of information. Self-report information solicited in formal questionnaires and obtained by interviewing and review of historical records needs to be considered in evaluating response bias and malingering of abilities. When available to evaluate the validity of self-report, psychometric instruments that provide quantification of response validity relevant to the claimed disabilities are preferred. For example, a normal score on a validity scale created for detection of *feigned mental illness* is not necessarily informative regarding the credibility of cognitive or physical abilities; a normal score on a validity scale for detection of *feigned cognitive complaints* may be informative. That is, malingering of psychological conditions can occur independently of malingering of abilities, such as cognitive abilities (e.g., Nelson, Sweet, Berry, Bryant, & Granacher, 2007), which necessitates using appropriate assessment methods that can evaluate both, if psychological disorder *and* decreased ability claims are present.

Unlike psychometric indicators of invalid performance, objective standards for the evaluation of inconsistencies between the clinical presentation and evidence of capacities outside the clinical setting may not exist. In the absence of such standards, a determination that an inconsistency indicates invalidity should be cautious and conservative and in some cases may be left to the trier-of-fact. Inconsistencies in the self-report of individuals, including those related to (1) the severity of initial injury that increases across time, (2) inaccurate reporting of premorbid capacities or premorbid health, and (3) the evolution of clinical presentation across time when a static condition would be expected would raise suspicion of, but alone may not indicate, malingering.

In making related decisions, clinicians consider the known degree of self-report reliability in routine clinical settings, which is at times lacking. When feasible, it can be important to also consider collateral sources of information, to the degree

that the persons providing such information have access to the salient facts needed and are disinterested parties.

Documentation within reports of procedures used to assess abilities

Neuropsychologists routinely list in their reports the assessment methods and procedures, including standardized tests, that are utilized in their evaluations. Symptom validity measures and related procedures should also be listed in neuropsychological reports. Including the names of the symptom validity measures and embedded indicators on which opinions are based assists readers in understanding the bases of the conclusions and facilitates re-evaluations. Neuropsychologists use their own judgment when determining the degree of detail that is included in descriptions of validity measures, mindful of the importance of safeguarding specific information regarding these measures that would preclude valid use in the future, if specific information was to be disseminated to non-neuropsychologists.

Consideration of evaluation context

The base rate of negative response bias varies as a function of setting. Individuals presenting as litigants, defendants, or claimants in a criminal, civil, or disability proceeding or otherwise with motive to appear symptomatic (e.g., academic accommodations, drug seeking, excusing from military duty) show an increased risk (e.g., Greve et al., 2009; Mittenberg et al., 2002). For this reason, individuals seen in a forensic context should be given measures that will assist in identifying or ruling out response bias (cf. Bush et al., 2005), which is an expectation that has been supported for years by numerous forensic researchers and experts. Especially because research has shown repeatedly that experienced experts are inaccurate in identifying valid versus invalid ability performances from mere observation of behavior or test scores (e.g., Ekman, O'Sullivan, & Frank, 1999; Faust, 1995; Faust, Hart, Guilmette, & Arkes, 1988), for a clinician to choose not to use effort tests and embedded validity indicators requires a solid justification, especially within a forensic context. In fact, there is consensus that a *decision* not to use effort tests and embedded validity indicators would only rarely be justified (cf. Bush et al., 2005). It is more common to not be *able* to use such measures, such as when the evaluation is severely restricted in terms of time constraints or administrative prohibition (e.g., Social Security disability evaluation) or the individual being evaluated is not appropriate to be given such measures (e.g., severe and well-documented mental retardation precludes formal testing procedures). When multiple validity indicators are not or cannot be relied on, it is the clinician's responsibility to document the reasons and explicitly note any resulting limitations to interpretations of findings.

Response bias may occur in routine clinical and medical referrals, when no forensic context is evident. When clinicians are evaluating a *patient* who by virtue of claimed injuries is reasonably likely to become a *litigant* or *claimant*, the clinician

should consider the increased risk of insufficient effort and response bias and construct evaluations accordingly.

Attempts should be made to limit false positive identifications of response bias. More diagnostic certainty is required in contexts that involve higher relative costs for false positive errors (e.g., cases that potentially involve the death penalty). Toward this end, the clinician should be knowledgeable regarding the populations on which specific effort indicators were developed. To the extent that an examinee differs from test-normative and other comparison samples, the clinician should be appropriately cautious in drawing conclusions regarding the presence of response bias. When cultural, ethnic, and/or language factors are present that are known to affect the results of the instrument being used, clinicians need to adjust their thresholds of identifying insufficient effort and response bias accordingly (Salazar, Lu, Wen, & Boone, 2007). Although stand-alone effort tests have not generally been found to be impacted by age and education, at extremes of these variables, effort test failures have been known to occur (e.g., <third grade reading level on Green's Word Memory Test; Green & Flaro, 2003).

Consensus recommendations for practitioners related to assessment of abilities

The following points are viewed as important for neuropsychologists to consider when addressing response bias and malingering related to abilities:

- Use of psychometric indicators is the most valid approach to identifying neuropsychological response validity.
- Stand-alone effort measures and embedded validity indicators should both be employed.
- In their reports, neuropsychologists list the symptom validity measures and procedures that are utilized in evaluations. Clinicians explain the bases of their opinions to the extent required by the forensic context, while avoiding inclusion of specific information pertaining to these measures that could preclude valid future use.
- The evaluation of self-reported symptoms is best accomplished using psychometric instruments containing proven validity measures.
- Substantial inconsistencies between test data and "real-world" activities and between self-report and historical records should be considered. When integrating various sources of information, clinicians should be mindful of incomplete or false history, which when substantially present may reflect negative response bias.
- As risk relates to the setting in which the evaluation is taking place, clinicians should be mindful of the larger context of the evaluation and the potential for litigation to develop.
- As with all types of psychological assessment, neuropsychologists routinely are expected to encourage optimal effort as a means of attaining best performance.

- Substantial discrepancy between test results and those known to occur with the alleged medical or psychiatric disorder should raise concern regarding the presence of insufficient effort, response bias, and malingering.
- Because effort can vary during an evaluation, if possible clinicians should use multiple validity measures covering multiple domains distributed throughout the testing. If the circumstances are such that testing must be brief (e.g., Social Security disability evaluations), minimally, embedded effort indicators should be examined. When multiple validity indicators cannot be relied on, it is the clinician's responsibility to document the reasons and explicitly note the interpretive implications.
- As the number and extent of findings consistent with the absence or presence of response bias increases, confidence in conclusions regarding the validity of the examination is strengthened accordingly.
- Clinicians should be cognizant regarding when examinee characteristics do not match those of effort test-normative and comparison samples, and should adjust interpretations and choose measures accordingly.
- When a psychological disorder (e.g., depression) *and* ability deficits (e.g., memory) are claimed, clinicians should administer measures that can evaluate response bias related to both.
- Serial evaluations can be particularly helpful in discriminating between genuine injury and unrealistic performances or variable self-report of deficits and disabilities that reflect variable effort and/or response bias.

Consensus recommendations for future scientific investigation related to assessment of abilities

As with all other scientifically based practice specialties, there are ongoing research efforts that strive to improve the effectiveness of procedures used by neuropsychologists. The following topics are viewed as important and desirable to pursue in terms of gathering additional scientific knowledge relative to the assessment of response bias and malingering of abilities:

- Additional research is needed regarding the manner in which "weighting" or "aggregation" of cognitive effort measures and embedded validity indicators of ability measures is best accomplished in clinical practice.
- Populations at risk of failing effort and embedded validity indicators despite best effort should be investigated.
- Additional methods should be developed that will provide evidence of *deliberate intent* to feign.
- New ability tests should have validity indicators created at the time of test construction.
- Cost–benefit of response bias assessment and malingering detection methods should be analyzed.
- Effort measures and embedded validity indicators should be applied to pediatric samples.
- Identification of the point at which risk of response bias has effectively been ruled out should be investigated.

SOMATIC ISSUES

Conceptual and operational definitions

Response validity regarding somatic symptoms relates to excessive subjective disability attributed to somatic dysfunction, which may result from claims of injury or illness. Examples include claims of physical symptoms purported to be associated with fibromyalgia, chronic fatigue syndrome, multiple chemical sensitivity, and other medically unexplained conditions (Binder & Campbell, 2004), in which exaggeration and/or fabrication of symptoms and disability is suspected to play a role.

Somatic complaints may be either specific or nonspecific in nature. Specific complaints may include complaints of focal weakness or paresthesia, lateralized sensorimotor impairment, and primary sensory changes (e.g., loss or reduction of vision, smell, or other sensory abilities). Non-specific complaints may include general malaise, fatigue, generalized weakness, dizziness, balance problems or vague generalized complaints of "tingling" or numbness. A primary domain of somatic complaints is pain (e.g., headache, low back pain, neck pain), which may be specific or non-specific. Pain symptoms may vary in terms of the qualitative nature of the pain, as well as pain severity, and the subjective report of pain-related disability (Bianchini et al., 2005).

Non-credible somatic symptoms may present as exaggerated or atypical symptom report on general personality tests, or exaggerated or atypical symptom report on specialized rating scales. Persons complaining of non-credible somatic symptoms may under-perform on measures of strength/dexterity, and may also show evidence of impaired cognitive performance that is atypical for bona fide clinical disorders. Evidence of non-credible somatic disability presentation shows base rates of 30–40% in secondary gain contexts (Greve, Ord, Bianchini, & Curtis, 2009; Mittenberg et al., 2002; Meyers, Millis, & Volkert, 2002) similar to those reported for non-credible cognitive performance disability.

Methods of assessment

Self-report. As noted in the Ability section of this consensus statement, it is important to assess the reliability and validity of examinee's self-report that is obtained by interviewing. Comparison of an examinee's self-reported history with available information from other reliable sources can assist in determining whether or not history is accurate and symptoms are consistently reported. Clinicians should be mindful of the known tendency among some forensic examinees to misrepresent (usually in a positive direction) their pre-incident historical status, in terms of cognitive, somatic, and/or psychological function (e.g., Gunstad & Suhr, 2001; Mittenberg, DiGuilio, Perrin, & Bass, 1992).

Extended personality inventories—e.g., Minnesota Multiphasic Personality Inventory-2 (MMPI-2); Personality Assessment Inventory (PAI)—as well as other more focused pain scales—e.g., Pain Patient Profile (P3)—are relevant to the assessment of potentially over-reported somatic symptoms. Such inventories typically show the greatest exaggeration on scales related to somatic symptoms,

with additional exaggeration, to a smaller degree, on scales measuring symptoms of depression and anxiety (Larrabee, 1998, 2007). Specialized validity scales, such as the MMPI-2 FBS (Symptom Validity scale) of the MMPI-2, have shown good discriminative validity and classification accuracy in the detection of exaggerated somatic symptoms (Greiffenstein, Fox, & Lees-Haley, 2007; Nelson, Sweet, & Demakis, 2006). Newer scales, (e.g., Fs; Ben-Porath & Tellegen, 2008), specifically tailored to somatic over-reporting have also been developed and may show merit in detecting exaggerated symptom presentation.

Motor skills. Unlike self-report, examination of motor skill performance in non-credible somatic symptom report is less studied. However, select investigations suggest potential utility of motor skill assessment in the detection of feigned somatic presentations. Finger-tapping scores among compensation-seeking individuals with independent evidence of non-credible cognitive performance fall substantially below the clinical benchmarks relative to non-compensation-seeking *patients* with genuine neurological injury (Arnold et al., 2005; Larrabee, 2003). Abnormally poor performance on a grip strength device can also provide evidence of feigned motor impairment (Greiffenstein, 2007). Last, feigned motor impairment can occur in disease-deficit-incompatible patterns. For instance, poor performance on gross relative to fine motor tasks is a pattern opposite to that produced by patients with documented neurologically based motor dysfunction (e.g., Greiffenstein, Baker, & Gola, 1996). Clinical benchmarks of motor performance among patients with known motor dysfunction (e.g., Butters, Goldstein, Allen, & Shemansky, 1998; Greiffenstein, 2007) should be considered.

Sensory/perceptual. A number of authors (Binder, Kindermann, Heaton, & Salinsky, 1998; Binder, Salinksy, & Smith, 1994; Mittenberg, Rotholc, Russell, & Heilbronner, 1996; Trueblood & Schmidt, 1993) have summarized difficulties in evaluating finger localization and graphesthesia in the context of non-credible impairment of sensoriperceptual function. In select cases that involve feigned primary sensory impairment, customized forced-choice measures can inform assessment of non-credible presentation (Pankratz, 1979).

Symptom validity tests. Patients exaggerating somatic symptoms may also fail standard cognitive symptom validity tests, also referred to as effort tests. In fact research has shown that base rates of symptom validity test failure are increased among compensation-seeking individuals with conditions, such as fibromyalgia (Gervais et al., 2001), toxic exposure (van Hout, Schmand, Wekking, Hageman, & Deelman, 2003), and chronic pain (Meyers & Diep, 2000; Meyers & Volbrecht, 2003). Legitimate somatic discomfort does not lead to patterns of suspected feigned cognitive impairment (Etherton, Bianchini, Greve, & Ciota, 2005). For these reasons, inclusion of cognitive symptom validity tests (forced-choice or non-forced-choice approaches) should be strongly considered. See the Ability Issues section of this statement for additional discussion of symptom validity tests.

Salient variables

Demand characteristics of a given response validity measure vary according to the nature of the external incentive. For instance, consistent with the scale's original

development in 1991, FBS appears more relevant to civil settings (Nelson et al., 2006) because over-reporting of severe psychiatric symptoms is more characteristic of individuals evaluated in criminal settings (Wygant et al., 2007).

Response validity on many ability measures does not relate to gender. However, a subset (e.g., response validity of motor performance; Arnold et al., 2005) has been found to benefit from gender-based cut scores. Similarly, assessing the validity of symptom reporting for some methods may benefit from consideration of gender, whereas other methods may not benefit from analysis of this or other moderator variables. There are limited data with regard to the effect of moderator variables on other validity scales and symptom validity tests designed to evaluate the accuracy of somatic symptom complaints.

Consensus recommendations for practitioners related to assessment of somatic symptoms

The following points are viewed as important for neuropsychologists to consider when addressing response bias and malingering of somatic presentation:

- When assessing for non-credible somatic presentation, use multiple well-validated measures covering domains of self-report, performance, and symptom validity. As the number and extent of findings consistent with absence or presence of response bias increase, confidence in conclusions regarding the validity of the examination is strengthened accordingly.
- Carefully rule out plausible alternative explanations, other than malingering, for the somatic presentation, as it is critically important to keep false positives to a minimum. The clinician is encouraged to consider actuarial data along with clinical judgment of patient self-report when making determinations of veracity of somatic complaints.
- Related to assessing the validity of somatic complaints, history information should be evaluated for completeness and accuracy.
- Keep current with literature that addresses non-credible somatic presentation.

Consensus recommendations for future scientific investigation related to Assessment of somatic symptoms

The following points are viewed as important to consider when addressing response bias and malingering of somatic presentation:

- It is essential that researchers seriously consider the *criterion problem*. That is, researchers must form criterion groups of ''malingerers'' independently of dependent variables.
- It is recommended that researchers employ stringent inclusion/exclusion criteria in their investigations. Control or comparison groups should consist of medical patients without known external incentives. The ideal criterion group is one that has (a) little to no clinical or laboratory evidence for pathology, combined with (b) symptom histories that are compellingly illogical to a reasonable clinician.

- Many past investigations have employed, as a comparison group, patients who themselves are seeking or receiving compensation, without any screening for symptom exaggeration or invalid test performance. Should an investigator wish to examine medical patients in whom external incentive is a factor, comparison group participants should be carefully screened a priori for the presence of non-credible somatic symptoms.
- In conjunction with other more commonly employed approaches, future studies might consider use of alternative functional capacity measures (e.g., Waddell signs; Waddell, McCulloch, Kummel, & Venner, 1980) as converging supportive evidence of exaggerated somatic presentation. Colleagues from other disciplines, such as medicine, might collaborate in establishing optimal classification accuracy results in such areas as functional capacity assessment, although neuropsychological researchers will need to appreciate limitations and differences in these approaches as compared to more typical exaggeration and comparison groups.
- The influence of demographic variables, such as gender, should continue to be a focus of investigation.

PSYCHOLOGICAL ISSUES

Conceptual and operational definitions

"Psychological issues" refers to those psychological/psychiatric disorders or conditions that may be seen in evaluations of claimants in secondary gain contexts. Typical contexts may include Independent Medical Examinations (IMEs) referred by insurance carriers, personal injury cases in civil litigation, disability evaluations for workers' compensation and Social Security, Department of Veterans Affairs claims for disability compensation, criminal prosecution for competency to proceed, the insanity defense/diminished capacity, or mitigation of the death penalty, among others (Boyd, McLearen, Meyer, & Denney, 2007; Denney, 2007, 2008; Greiffenstein, 2007; Rogers, 2008).

In this section we discuss response validity assessment in claims of psychopathology and/or emotional distress, specifically in the form of exaggeration, symptom promotion, or frank symptom fabrication. Common disorders primarily include, but are not limited to, anxiety disorders, depression and related disorders (e.g., bipolar disorder), psychoses, and post-traumatic stress disorder (PTSD). The committee recognizes that PTSD is classified as a type of anxiety disorder (APA, 2000), but chose to represent it as a separate diagnostic entity within this consensus statement because of its prevalence in disability claims, especially within military settings and the Department of Veterans Affairs, and also because of the presence of unique attributes of the disorder (i.e., the etiology is dependent on a traumatic stressor; APA, 2000). The current list is not intended to be exhaustive. Other common disorders may also be encountered in secondary gain examination contexts, such as attention deficit hyperactivity disorder (ADHD) and/or learning disability (LD) within an assessment context for test-taking accommodations (Mapou, 2008; Osmon & Mano, 2009). Some disability insurance carriers may also compensate individuals with substance abuse disorders.

Diagnostic considerations

Regardless of the specific type of psychopathology, it is incumbent upon the examiner to pursue a formal, in-depth diagnostic evaluation. It is the *totality* of the claimant's presentation that should be taken into account when assessing the validity of claims of psychopathology and/or emotional distress. Neuropsychologists are well suited to this endeavor by virtue of their scientific training and experience in psychopathology, knowledge of differential diagnosis, expertise in psychometrics, and skills in the assessment of symptom validity and response bias. In fact, neuropsychologists have largely been responsible for the development and growth of this important area (Boone, 2007; Larrabee et al., 2007; Sweet, 2009; Sweet, Ecklund-Johnson, & Malina, 2008). Examiners must familiarize themselves with the discrete diagnostic criteria of the condition in question with the most current authoritative diagnostic sources. Specific attention should be directed toward the most common features of the disorder in question, including the onset, course, symptom picture, co-morbidities, treatment efforts, and response to treatment. With these diagnostic criteria in mind, the examiner can address whether the claimant's presentation is, or is not, compatible with what is known about the disorder. The presence of inconsistencies and contradictory data should be noted and documented.

Diagnostic considerations necessarily take into account the following information.

Onset. The compatibility of disorder onset with the general principles of what is known about the particular psychological/psychiatric condition in question is important. In this regard, the examiner may note obvious inconsistencies, such as the claim of ''schizophrenia'' with onset well past the typical age of onset. In criminal contexts, in individuals with no psychiatric history, observations of first symptom onset *after the defendant's arrest* should raise suspicion. The examiner must determine whether the onset of the disorder makes sense and is consistent with well-established knowledge of psychopathology.

Similarly, elucidation of the *claimant's and his/her family history* can be informative. Again, as stated in earlier sections of this consensus statement, self-report information obtained by interviewing needs to be evaluated for accuracy. Some early childhood circumstances (e.g., abuse, neglect) have the potential to predispose an individual to the development of psychopathology later in life (Grover et al., 2007; Molnar, Buka, & Kessler, 2001). Although the lack of a family history of psychopathology may not undermine the credibility of claimant's presentation, the presence of a positive family psychiatric history may ultimately be helpful in differential diagnoses.

Symptom presentation. The consistency of reported and observed symptoms with what is typically expected in the disorder can be informative. The presence of atypical/unusual symptoms, such as odd hallucinations or those that appear to represent a layman's conception of the disorder, may raise suspicion of symptom fabrication. The presence of an unusual combination of symptoms (e.g., a claimant who experiences hallucinations only in the presence of his/her pet dog) may suggest symptom fabrication (Guy, Kwartner, & Miller, 2006).

Course. The degree to which symptom presentation follows a course or pattern over time that is typical or atypical of the disorder at issue can be revealing. For example, a claimant purporting to have auditory hallucinations 24 hours a day (i.e., without even brief periods of remission) or the claimant who is catatonically depressed at a given time only to be spontaneous and conversant the next moment may raise questions regarding validity of symptoms.

Treatment/response to treatment. An examinee's documented history may indicate that the claimant has been formally diagnosed with the disorder in question by an appropriately credentialed mental health professional. This documented history within the examinee's records conceivably may provide some historical evidence of credibility. However, the accuracy and ultimate credibility of that diagnosis should be viewed with an appropriate degree of skepticism and confirmed within the broad and comprehensive scope of the present examination, rather than necessarily be taken at face value. Thus, the possibility of incorrect prior diagnoses should be carefully explored. It is important to note whether the diagnosis was made in a clinical context apart from a secondary gain context. Has the examinee sought appropriate treatment for the disorder? Has he/she responded as expected to treatment efforts or does he/she remain largely refractory? In this regard, there is little doubt of the meaningfulness of the situation when an examinee remains refractory to treatment for depression only *after* application for compensation.

Assessment methodology

General considerations. The assessment of psychological and psychiatric issues by neuropsychologists is a data-driven enterprise, which may best be characterized as a search for "consistencies and inconsistencies." Operationalizing this concept, the examiner strives to answer the question, "Does what I am learning about this claimant make sense in light of the putative claim, the diagnosis, history and totality of the presentation?" Assessment is a multi-faceted endeavor requiring the integration of numerous discrete data sources, typically including: (1) review of records; (2) psychosocial history obtained by interviewing; (3) observations of the claimant's behavior during the assessment period; (4) consideration of information from collateral sources, such as significant others, employers, etc., when available and appropriate; (5) formal psychological/neuropsychological testing; (6) response validity assessment procedures; and (7) surveillance video/audio, when available. Examiners are encouraged to utilize best practices and follow current practice guidelines (e.g., AACN, 2007).

When feasible, it is often appropriate to obtain all available medical, psychiatric, legal, and other relevant records. Careful scrutiny of records, with particular attention to premorbid status, reported onset and course of symptoms, treatment efforts, and response to treatment can be informative. This review may reveal the presence of contradictory and/or confirmatory opinions of other professionals, which warrant consideration.

The examiner's interview of the claimant will necessarily be thorough, with particular attention paid to possible inconsistencies in behavior, demeanor,

symptoms, etc. Is the claimant's presentation consistent with the reported diagnosis and aspects of the history? Does his or her mental status make sense in light of reported symptoms and history, or does it stand at odds with all or part of it? Is the onset of the disorder plausible? Some examinees, particularly in criminal forensic evaluative settings, may be so naïve and unsophisticated as to "develop" symptoms after their arrest (Morgan, 2008). While most claimants are likely to be more sophisticated than that, the examiner should be sensitive to such temporal factors.

Historical information can be relevant to all forms of psychopathology, particularly with regard to post-traumatic stress disorder (PTSD), as the nature and extent of the trauma allegedly experienced by the claimant is crucial in determining a plausible etiology and diagnosis of the disorder. Examiners attempt to inquire about all aspects of the alleged stressful event(s) in question, the context of occurrence, and with special inquiry into its plausibility. Some claimants may present a history in which they did not experience an event that "involved actual or threatened death or serious injury, or threat to the physical integrity of self or others" (APA, 2000, p. 467). Claimants may sometimes present plausible-sounding events, which may ultimately be shown to be fabricated or significantly embellished. A detailed history of pre- and post-injury life stressors should be explored with emotional stress claims.

Cultural, ethnic, and socio-economic factors should be taken into assessment and diagnostic considerations, as some forms and/or expressions of psychopathology may be varied or more prevalent among certain demographic groups (U.S. Department of Health & Human Services, 2000). Distinct clinical presentations and/or prevalence of psychopathology may differ among some cultures and ethnic groups (Draguns & Tanaka-Matsumi, 2003).

Consistency/inconsistency of examinee behavior over time can be important. This is within an evaluation and also true as it relates to an examinee's behavior outside of the test environment, which might be revealed via video surveillance. When present, the presence of unusual symptoms, an irregular pattern or course of the disorder, vague or odd onset, non-credible stressors, atypical response to treatment efforts, a waxing-waning/remitting course, etc, need to be considered. Collateral information can reveal consistency or inconsistency in history and presentation and should be obtained and reviewed when possible. Such information may confirm or fail to confirm the claimant's self-report.

Psychological and neuropsychological assessment

Because many claims regarding psychiatric diagnoses involve claimant reports of impaired cognitive functions (ostensibly related to the psychiatric condition), comprehensive neuropsychological assessment is frequently warranted. Most claims related to psychological/psychiatric disorders include alleged impairments in attention, processing speed, memory, and executive functions. Examinees may report cognitive dysfunction in the area of attention deficits and/or memory impairment reportedly related to anxiety, depression, or some other psychiatric disorder. In addition, some symptoms that primarily present as a neurocognitive disorder are believed to have their basis in psychopathology (e.g., psychogenic non-epileptic seizures, PNES; Alper, 1994; Reuber & Elger, 2003), thus seemingly

clouding the sometimes artificial distinction between cognitive and psychiatric disorders and symptoms. As with any cognitive assessment, particularly in a secondary gain context, formal tests of cognitive and emotional symptom validity are indicated. Examiners are advised not to rely on a single symptom validity test, but multiple measures administered throughout the assessment day(s) are suggested. Such measures may be useful to assist in the determination of non-credible claims of emotional distress, exaggerated claims of cognitive dysfunction and/or poor effort. See the Ability Issues and Somatic Issues section of this statement for additional discussion on symptom validity tests.

In the assessment of exaggerated symptoms of emotional/psychiatric distress and the detection of feigned psychological symptoms, a number of freestanding measures are available for various conditions (e.g., PTSD, depression). Multi-scale inventories of psychopathology (e.g., MMPI-2) are well known to neuropsychologists. Many of these include specific scales and items designed to assess the validity of complaints of psychiatric and emotional symptoms. An example of this type of measure is the Structured Interview of Reported Symptoms (SIRS).

Clinicians need to stay current with the relevant peer-reviewed literature in order to determine whether to rely on such scales. Use of multiple indices of both cognitive and emotional validity in assessments can insure that the diagnostic process is as accurate as possible.

Neuropsychologists, by virtue of their training, clinical judgment, and experience, and informed by well-validated assessment methods, knowledge of the condition in question, and relevant scientific literature, can identify the presence and severity of genuine psychopathology. The presence of symptom exaggeration, response bias, and/or symptom fabrication can be determined by the presence of a combination of factors, including non-credible/invalid response patterns, non-credible history/presentation, and or lack of corroboration by collateral data sources and/or medical records.

A determination of malingered psychopathology, whether exaggerated or wholly feigned, can be made on the basis of self-report, psychological/neuropsychological test results, and observations/ corroborative information. When claims of emotional distress are disproportionate to the history provided by the claimant and other sources of information, and/or when inconsistencies in self-report over time and across evaluations are present, and/or when symptom reports do not follow what is known about the progression of symptoms and the natural history of the claimed disorder, and/or when symptom presentation is atypical of the disorder, a determination of exaggerated or feigned psychopathology may be justified. It is important to recognize that even in cases of well-confirmed, genuine psychopathology, malingering may co-exist. Indeed, genuine psychopathology and symptom fabrication are not mutually exclusive (Morgan & Gervais, 2009; Morgan, Millis, & Mesnik, 2009; Slick et al., 1999). However, when neuropsychological and psychological test results reveal multiple indicators of invalid test performance, even in the context of genuine psychopathology, there is an increased possibility that exaggerated and or feigned/malingered psychopathology is present.

Consensus recommendations for practitioners related to assessment of psychological symptoms

The following points are viewed as important for neuropsychologists to consider when addressing response bias and malingering of psychological symptoms of psychological presentation:

- Clinicians engaged in assessment of an individual's psychological issues within a forensic context need to be aware that self-report may be biased, false, or incomplete, and proactively evaluate this possibility.
- For individuals undergoing forensic assessment, both cognitive and/or emotional complaints are common co-occurrences in this population, requiring that both domains be appropriately assessed and the validity of each determined.
- Clinicians would benefit from establishing their own practice-specific database on examinees, consisting of in-depth interview and complete history, observations of behavior, informant/collateral interviews, and formal neuropsychological/psychological testing.
- Assessment instruments and scales should be utilized that provide the most current, scientifically informed methodology for the assessment of emotional/psychopathology and cognitive factors.
- Clinicians should utilize multiple symptom validity measures administered throughout the evaluation. Confidence in conclusions is expected to be commensurate with the number and extent of findings demonstrating absence or presence of response bias.
- Because the co-occurrence of genuine psychopathology and feigned/exaggerated symptoms is common, examiners should, as much as possible, attempt to delineate the relative presence of each. Examiners should be familiar with base rates of mental disorders and symptoms in the general population.
- Clinicians are encouraged to use best clinical practices, assessment methodologies (instruments, scales, scoring criteria, etc.) that are current, and be current consumers of the relevant scientific literature.
- Because research and clinical experience indicate that some cultural and ethnic differences exist with regard to the presentation of psychopathology, clinicians are encouraged to consider such factors, as appropriate to the individual case.

Consensus recommendations for future scientific investigation related to assessment of psychological symptoms

The following points are viewed as important for neuropsychologists to consider when addressing response bias and malingering:

- Researchers are encouraged to continue to investigate emotional and cognitive response validity issues in known psychiatric groups and to continue the development of new instruments and scales to detect response bias.
- The relationship between genuine psychopathology and invalid response patterns requires ongoing empirical investigation and scientists are encouraged to work toward better elucidation of this relationship.

- Scientists engaged in personality/psychopathology research are encouraged to become more familiar with invalid self-report and attempt to understand those individual differences that may contribute to distortion, exaggeration, or feigning of self-report.

RESEARCH EVIDENCE AND SCIENTIFIC ISSUES

Research designs

Three research designs that have been most commonly used in effort-testing studies are: (a) simulation; (b) criterion groups (''known'' groups); and (c) differential prevalence designs. The reader is referred to Rogers (2008) for a detailed description and analysis of these different designs.

In analog simulation studies, non-clinical participants, such as community volunteers or college students, are assigned to different experimental conditions (e.g., given instructions to simulate) and compared to clinical samples of participants with verified cognitive impairment on the test of interest. This analog design often provides tight experimental control as well as a practical and cost-effective method for examining ''proof of concept'' for new tests. A major limitation of this design is one of generalizability (i.e., that the behavior elicited may be different than behavior encountered in the real world; Rogers, 2008).

A criterion (''known groups) design uses a priori criteria to define response bias, such as selecting people in litigation for mild brain injury claims who perform at chance level on a forced-choice test. Criteria typically are chosen that optimize a very low false positive rate. As with the analog simulation design, the criterion group's performance on the test of interest is compared to a clinical group's performance. The criterion group design has the strength of clinical relevance by including people who have real-world incentives to malinger. However, developing appropriate external criteria for defining response bias can be a major methodological challenge.

The differential prevalence design assumes that response bias prevalence varies as a function of environmental context and likelihood of incentives to malinger. For example, it is inferred that response bias is more common in persons who are litigating versus those who are not. Groups are composed on the basis of assumed incentives. Use of differential prevalence designs may be most useful in initial validation studies. A major limitation of the differential prevalence design is that it is difficult, if not impossible, to know the accuracy of the inferences made.

It is the consensus of this panel that the simulation and criterion groups designs represent rigorous and clinically relevant research designs. The differential prevalence design can yield additional information relevant to test development, but it should not be used as the sole or primary research design for test validation. In general, researchers are encouraged to use multiple designs during test development and validation. In addition, it is important to include appropriate clinical groups in a systematic program of research that is designed to examine a test's construct and predictive validity.

Statistical and methodological issues

Single versus multiple indicators. Of the various types of tests designed to detect response bias, single, *stand alone* forced-choice tests have been the best validated. These single tests will continue to be useful in neuropsychological assessment. However, sole reliance on single tests raises diagnostic concerns. The extent to which single tests are vulnerable to detection, disclosure, and coaching will determine whether a particular test will lose diagnostic sensitivity to response bias (Horwitz & McCaffrey, 2006; Youngjohn, 1995). In addition, there is the question of whether the detection of response bias can be improved incrementally beyond the use of single tests. Researchers have encouraged the use of multiple measures as a method for increasing classification accuracy (Larrabee, 2003; Nelson et al., 2003; Victor, Boone, Serpa, Buehler, & Ziegler, 2008).

The panel recommends more research on methods that combine multiple stand-alone tests and/or embedded measures. Common methods include unit weighting (Dawes, 1979) and logistic regression (Menard, 2002). Multivariable test composites may be more resistant to coaching, especially when based on standard neuropsychological tests. In addition, methods like logistic regression can assist in determining which tests or variables produce incremental power in differentiating groups, and estimate optimal weights for the tests, while controlling for test redundancy. Simple unit weighting has been criticized for failing to account for the dependencies among tests, which likely results in overly optimistic predictions (Chan, Deeks, Macaskill, & Irwig, 2008). However, in their review of the literature, Bobko, Roth, and Buster (2007) found that unit weighting has substantial predictive validity. Larrabee (2008) demonstrated the usefulness of a unit-weighting method in the symptom validity context.

Although multivariable composites offer considerable potential in deriving new tests and embedded measures, there are certain statistical pitfalls that need to be avoided. When developing multivariable models, investigators must select a set of predictor variables. It is not uncommon to have dozens of variables from which to choose. However, one risks overfitting the model if there are not at least 10 to 20 participants for every predictor variable in the regression context. In the case of binary logistic regression, this estimate is based on the sample size of the smaller of the two groups, not the total sample (Harrell, 2001). Overfitting will exaggerate the predictive worth of the model. The chance of finding spurious associations between the dependent variable and predictor variables will be greatly increased. Some investigators have looked to stepwise variable selection to solve the problem of too many variables. Stepwise variable selection includes a variety of methods for selecting subsets of variables on the basis of the change in the residual sum of squares as a result of including or excluding the variable from the model. However, Harrell (2001) has noted that: (a) stepwise techniques produce biased standard errors and inflated R^2 values, (b) the F and χ^2 test statistics reported by stepwise methods do not have the claimed distributions, (c) the reported p values are too small; (d) multicollinearity among the predictor variables greatly affects which variables are selected by stepwise methods; and (e) the final model will often contain variables that are not accurate predictors of the dependent variable.

Harrell (2001) provides detailed strategies for variable selection. First, research hypotheses, theory, and past research findings should still guide variable selection. Newly developed statistical techniques such as Bayesian model averaging (Wang, Zhang, & Bakhai, 2004) and penalized maximum likelihood estimation (Moons, Donders, Steyerberg, & Harrell, 2004) represent different, but useful approaches, to the variable selection problem.

Whether using a multivariable composite or a single test, neuropsychologists should not rely on single, fixed cut scores. Neuropsychologists appreciate and consider a range of cut scores and associated diagnostic test statistics in choosing the cut score to be applied to a specific case. The decision-making process occurs in different contexts, such that the relative costs of false positive and false negative errors will not be constant across situations. Raising or lowering a test's cut score will increase or decrease the test's sensitivity and specificity in an inverse fashion: when sensitivity is increased, specificity decreases. To assist clinicians in this decision-making process, investigators, journal editors, and test publishers are strongly encouraged to provide a broad range of cut scores with their respective diagnostic test statistics (e.g., sensitivities, specificities, and likelihood ratios).

All tests and score combinations require validation. There are two major methods of model validation: external and internal (Harrell, 2001). External validation generally involves an independent validation sample with participants from a different, but similar, population (Justice, Covinsky, & Berlin, 1999). Although ideal, external validation can be costly in terms of time and resources. Some investigators will split their original sample into derivation and validation samples. However, this procedure is problematic because it can result in lower precision and power (Harrell, 2001). Internal validation procedures, such as bootstrapping (Steyerberg, Bleeker, Moll, Grobbee, & Moons, 2003; Steyerberg et al., 2001), offer a useful supplement to external validation and do not require sample splitting. The basic concept of bootstrapping involves drawing samples (e.g., 500) with replacement from the original data set of the same size, computing the statistic of interest, and examining how the statistic changes over the 500 repetitions. This process allows the investigators to determine the degree of "optimism" in the original model.

Diagnostic statistics. Differences on test scores between two groups are commonly analyzed statistically with the *t*-test or the Mann-Whitney-Wilcoxon test. Although these tests are useful, they are insufficient to evaluate the performance of tests and embedded measures. Researchers and clinicians need to have a working knowledge of common diagnostic statistics, including but not necessarily limited to, those listed below (Straus, Richardson, Glasziou, & Haynes, 2005).

- Sensitivity: The proportion of persons with the disorder (or condition or behavior of interest) who obtain a positive score on the effort test (i.e., fail the test).
- Specificity: The proportion of persons without the disorder (or without the condition or behavior of interest) who obtain a negative score on the effort test (i.e., pass the effort test).
- Positive predictive value (PPV): The proportion of patients with positive test results who are correctly classified. It should be noted that PPV, unlike

sensitivity and specifity, varies as a function of the prevalence (i.e., base rate) of the disorder (or condition or behavior of interest).

$$PPV = \frac{(sensitivity)(prevalence)}{(sensitivity)(prevalence) + (1 - specificity)(1 - prevalence)}$$

- Negative predicted value (NPV): The proportion of patients with negative test results who are correctly classified. NPV also varies as a function of disorder prevalence.

$$NPV = \frac{(\text{specificity})\,(\text{prevalence})}{(\text{specificity})(prevalence) + (1 - \text{sensitivity})(1 - \text{prevalence})}$$

- Likelihood ratio: [Sensitivity / (1 – Specificity)]. The percentage of people with the disorder with a positive test (i.e., true positives) divided by the percentage of people without the disorder who have a positive test results (i.e., false positives). It indicates how many times more (or less) likely it is that people with the disorder obtain a positive test result compared to those without the disorder who obtain a positive test result. A likelihood ratio greater than 1.0 indicates that the test result is associated with the presence of the disorder of interest. A likelihood ratio of less than 1.0 is associated with the absence of the disorder (Straus et al., 2005). Likelihood ratios from 2.0 to 5.0 yield small increases in the post-test probability, from 5.0 to 10.0 moderate increases, and above 10.0 large increases (Grimes & Schulz, 2005).
- Area under the curve (AUC): Area under the receiver operating characteristic (ROC) curve, or AUC. The ROC curve is a plot of a test's sensitivity (plotted on the y-axis) versus its false positive rate (i.e., 1 – specificity) plotted on its *x*-axis. Each point on the graph is generated by a different cut score on the test. Each point can be connected, a curve can be generated, and the area under the curve can be estimated parametrically (via maximum likelihood estimation) or non-parametrically (via the trapezoidal rule). The ROC curve area has several interpretations (Zhou, Obuchowski, & McClish, 2002): (a) the average value of sensitivity for all possible values of specificity, (b) the average value of specificity for all possible values of sensitivity, and (c) the probability that a randomly selected patient with the disorder has a test result indicating greater suspicion than that of a randomly selected patient without the disorder. Hosmer and Lemeshow (2000) offer some guidelines for interpreting the magnitude of AUC: (a) .50 = no discrimination; (b) .70 – .80 = acceptable discrimination; (c) .80 – .90 = excellent discrimination; and (d) ≥.90 = outstanding discrimination.

Reporting of diagnostic accuracy studies

It is recommended that investigators who are reporting the findings of diagnostic studies of SVTs follow the Standards for Reporting of Diagnostic

Accuracy (STARD) Guidelines (see Bossuyt et al., 2003). In 1999, the Cochrane Diagnostic and Screening Test Methods Working Group discussed the poor quality and substandard reporting of many diagnostic test evaluations. This working group then developed the STARD initiative. The STARD Guidelines are designed to improve the quality of the reporting of diagnostic studies. A flow diagram and spreadsheet are available to assist investigators in describing the important components of study design, method of participant recruitment, description of the reference and index tests, the conduct of the study, the administration of the tests, and the results. The diagram and checklist are available on the STARD website: http://www.stard-statement.org/. Neuropsychologists are encouraged to apply the STARD guidelines to evaluate diagnostic studies of embedded measures, stand-alone tests, and multivariable algorithms.

SUMMARY

Issues related to effort, response bias, and malingering have been prominent in the professional journals and at the professional meetings of clinical neuropsychologists for years. The detection of problematic effort and response bias, and the determination that malingering is present or absent, are important functions of clinicians, particularly because there is a substantial risk that such factors could be present among the numerous cases assessed by neuropsychologists in a secondary gain context. Even in a routine clinical context the presence of problematic effort and response bias can potentially invalidate results. The assessment of effort and genuine reporting of symptoms is important in all evaluations. The present Consensus Conference Statement is intended to assist clinicians and researchers, and should be viewed as reflecting current knowledge and available instruments. However, this area of investigation is by no means static. With the degree of energy and interest invested in these topics by clinicians and researchers, a dynamic and continued evolution of concepts, definitions, and related assessment procedures is to be expected. Practitioners are encouraged to remain informed regarding the local rules governing provision of expert opinions regarding response bias, effort, and malingering.

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APPENDIX: LIST OF PRE-CONFERENCE READINGS

General (for all groups to use)

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Heilbronner Working Group: Definitions and Differential Diagnosis

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Sweet Working Group: Ability Issues

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Larrabee Working Group: Somatic Issues

Bianchini, K. B., Greve, K. W., & Glynn, G. (2005). On the diagnosis of malingered pain-related disability: Lessons from cognitive malingering research. *Spine Journal*, *5*, 404–417.

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Morgan Working Group: Psychological Issues

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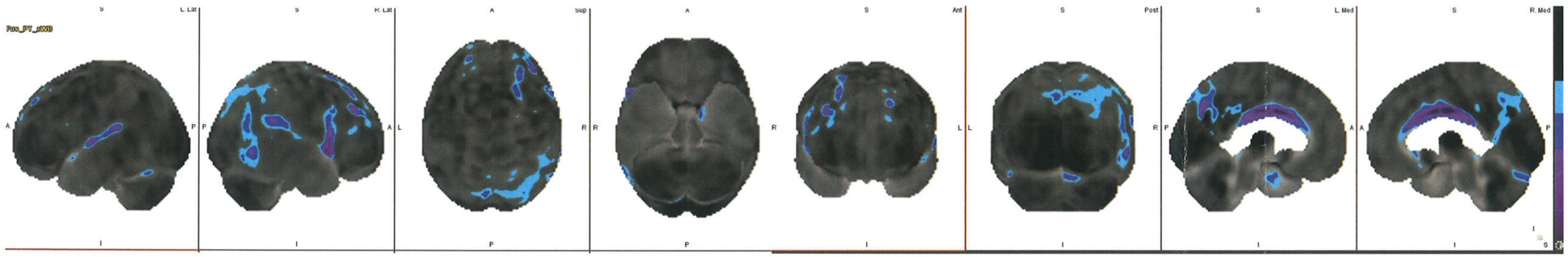
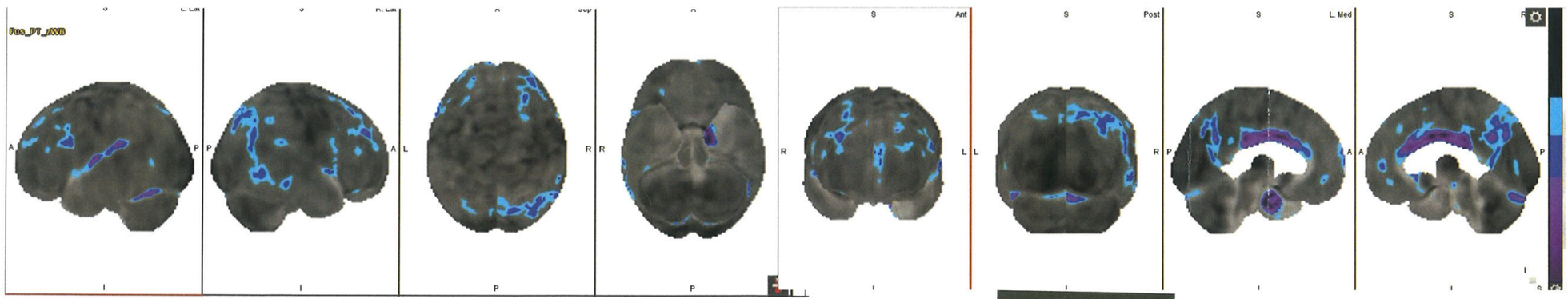
Gov. Ex. 170

Progression of FDG-PET



Brockman 3/12/21



Brockman 8/24/21

GOVERNMENT EXHIBIT
4:21-CR-009-GCH
No. 170

GOVERNMENT EXHIBIT
170

13

Gov. Ex. 171

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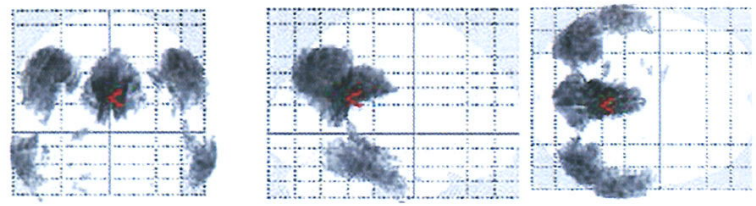
Dementia



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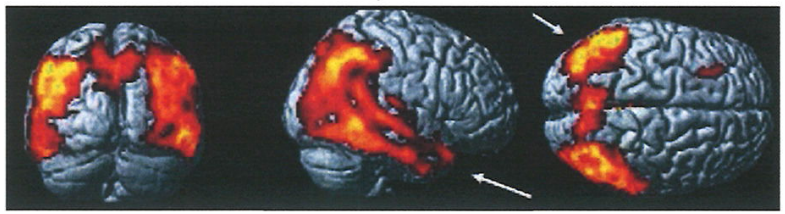

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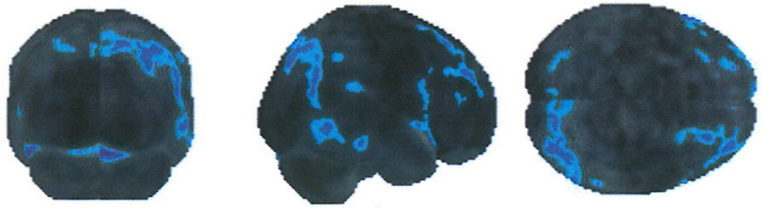

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Age-specific and sex-specific prevalence of cerebral β-amyloidosis, tauopathy, and neurodegeneration in cognitively unimpaired individuals aged 50–95 years: a cross-sectional study

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Summary

Background A new classification for biomarkers in Alzheimer's disease and cognitive ageing research is based on grouping the markers into three categories: amyloid deposition (A), tauopathy (T), and neurodegeneration or neuronal injury (N). Dichotomising these biomarkers as normal or abnormal results in eight possible profiles. We determined the clinical characteristics and prevalence of each ATN profile in cognitively unimpaired individuals aged 50 years and older.

Methods All participants were in the Mayo Clinic Study of Aging, a population-based study that uses a medical records linkage system to enumerate all individuals aged 50–89 years in Olmsted County, MN, USA. Potential participants are randomly selected, stratified by age and sex, and invited to participate in cognitive assessments; individuals without medical contraindications are invited to participate in brain imaging studies. Participants who were judged clinically as having no cognitive impairment and underwent multimodality imaging between Oct 11, 2006, and Oct 5, 2016, were included in the current study. Participants were classified as having normal (A–) or abnormal (A+) amyloid using amyloid PET, normal (T–) or abnormal (T+) tau using tau PET, and normal (N–) or abnormal (N+) neurodegeneration or neuronal injury using cortical thickness assessed by MRI. We used the cutoff points of standard uptake value ratio (SUVR) 1·42 (centiloid 19) for amyloid PET, 1·23 SUVR for tau PET, and 2·67 mm for MRI cortical thickness. Age-specific and sex-specific prevalences of the eight groups were determined using multinomial models combining data from 435 individuals with amyloid PET, tau PET, and MRI assessments, and 1113 individuals who underwent amyloid PET and MRI, but not tau PET imaging.

Findings The numbers of participants in each profile group were 165 A–T–N–, 35 A–T+N–, 63 A–T–N+, 19 A–T+N+, 44 A+T–N–, 25 A+T+N–, 35 A+T–N+, and 49 A+T+N+. Age differed by ATN group (p<0·0001), ranging from a median 58 years (IQR 55–64) in A–T–N– and 57 years (54–64) in A–T+N– to a median 80 years (75–84) in A+T–N+ and 79 years (73–87) in A+T+N+. The number of *APOE* ε4 carriers differed by ATN group (p=0·04), with carriers roughly twice as frequent in each A+ group versus the corresponding A– group. White matter hyperintensity volume (p<0·0001) and cognitive performance (p<0·0001) also differed by ATN group. Tau PET and neurodegeneration biomarkers were discordant in most individuals who would be categorised as stage 2 or 3 preclinical Alzheimer's disease (A+T+N–, A+T–N+, and A+T+N+; 86% at age 65 years and 51% at age 80 years) or with suspected non-Alzheimer's pathophysiology (A–T+N–, A–T–N+, and A–T+N+; 92% at age 65 years and 78% at age 80 years). From age 50 years, A–T–N– prevalence declined and A+T+N+ and A–T+N+ prevalence increased. In both men and women, A–T–N– was the most prevalent until age late 70s. After about age 80 years, A+T+N+ was most prevalent. By age 85 years, more than 90% of men and women had one or more biomarker abnormalities.

Interpretation Biomarkers of fibrillar tau deposition can be included with those of β-amyloidosis and neurodegeneration or neuronal injury to more fully characterise the heterogeneous pathological profiles in the population. Both amyloid-dependent and amyloid-independent pathological profiles can be identified in the cognitively unimpaired population. The prevalence of each ATN group changed substantially with age, with progression towards more biomarker abnormalities among individuals who remained cognitively unimpaired.

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Introduction

Use of biomarkers as an aid to the diagnosis of Alzheimer's disease gained acceptance with the publication of the National Institute on Aging–Alzheimer's Association (NIA-AA) recommendations[1–4] and the International Working Group (IWG) criteria[5,6] for Alzheimer's disease. In the NIA-AA recommendations, biomarkers were divided into two classes: biomarkers of

Research in context

Evidence before this study

We searched PubMed with the terms "preclinical AD", "tau PET", and "amyloid PET" for English-language articles published from Jan 1, 2006, to Sept 1, 2016. Cognitively unimpaired cohorts have been studied using the National Institute on Aging–Alzheimer's Association (NIA-AA) staging recommendations plus the suspected non-Alzheimer's pathophysiology (SNAP) construct with the terms amyloid abnormal (A+), amyloid normal (A–), neurodegeration abnormal (N+), or neurodegeration normal (N–), resulting in four different biomarker categories: A–N–, A+N–, A–N+ (SNAP), or A+N+. Proportions of these four groups were roughly similar in many cohorts. The proportion of *APOE* ε4 carriers was greater in the A+N– and A+N+ groups than in A–N– or SNAP. Clinical and psychometric outcomes were uniformly worst in individuals classified as A+N+. These findings were largely the same whether biomarker categorisation was done using imaging or CSF. The NIA-AA staging plus SNAP construct has been useful because it has provided a common framework for different research groups to communicate their own findings.

Added value of this study

In retrospect, a weakness of the NIA-AA staging plus SNAP construct is the grouping of CSF phosphorylated tau into the same neurodegeneration or neuronal injury category with total tau, MRI, and ^{18}F-fluorodeoxyglucose (^{18}F-FDG)-PET. The ATN construct remedies this weakness and enables researchers to investigate multidomain biomarker associations for which the effects of tauopathy (defined by tau PET or CSF phosphorylated tau) and neurodegeneration or neuronal damage (defined by CSF total tau, MRI, or ^{18}F-FDG PET) are segregated at an individual level. We described clinical characteristics and age-specific and sex-specific prevalence of amyloid, tau, and neurodegeneration or neuronal injury in individuals aged 50 years and older using the ATN construct. To our knowledge, this is the first study to do so. We showed that tau and neurodegeneration were often discordant: among individuals who would have been classified as NIA-AA stage 2 or 3 preclinical Alzheimer's disease (ie, A+T+N–, A+T–N+, and A+T+N+), tau PET and neurodegeneration biomarkers were discordant in 86% of those aged 65 years and 51% of those aged 80 years; among the individuals who would have been labelled SNAP (ie, A–T+N–, A–T–N+, A–T+N+) tau PET and neurodegeneration biomarkers were discordant in 92% of those aged 65 years and 78% of those aged 80 years.

Implications of all the available evidence

The ATN classification scheme is a useful approach to characterise abnormalities in the biomarkers of amyloid, tau, and neurodegeneration or neuronal injury to understand the underlying heterogeneous pathological profiles in the population. Marked age variation in biomarker prevalence requires careful interpretation of biomarker results from studies across cohorts of different ages. Future research will determine the within-individual biomarker changes to assess amyloid-dependent (ie, Alzheimer's disease) and amyloid-independent (ie, SNAP) pathological pathways and sequences of biomarker abnormality.

amyloid (A) and biomarkers of tau-related neurodegeneration or neuronal injury (N),[1–4] for which biomarkers were used to classify individuals as amyloid abnormal (A+), amyloid normal (A–), neurodegeration abnormal (N+), or neurodegeration normal (N–). When the NIA-AA recommendations for preclinical Alzheimer's disease staging were applied to a cohort of 450 cognitively unimpaired individuals aged older than 70 years, roughly a third were categorised as stages 1–3 preclinical Alzheimer's disease, 40% were amyloid normal and neurodegeneration normal (A–N–), and a quarter were amyloid normal and neurodegeneration abnormal (A–N+).[7] We labelled the A–N+ group as suspected non-Alzheimer's pathophysiology (SNAP) on the assumption that this was a pathologically heterogeneous group with various non-Alzheimer's pathologies.[7] To reflect NIA-AA staging while accounting for the SNAP and A–N– groups, many research groups have adopted a two-class biomarker construct in which individuals are assigned to one of four biomarker categories: A–N–, A+N–, A–N+ (SNAP), or A+N+.[8–13] This approach has been useful because it has provided a common framework for different research groups to communicate findings in their own cohorts.[8–13]

However, a weakness of the NIA-AA staging method plus SNAP construct was the grouping of CSF phosphorylated tau, MRI, and ^{18}F-fluorodeoxyglucose (^{18}F-FDG)-PET into one neurodegeneration or neuronal injury category.[1–4,7] In individuals with Alzheimer's disease, it is reasonable to assume that neurodegeneration in areas sensitive to Alzheimer's disease is most often related to tauopathy; however, neurodegeneration, even when defined on the basis of its pattern in Alzheimer's disease, also occurs in disorders other than Alzheimer's disease. A solution to this problem is to separate biomarkers that are specific for deposits of fibrillar tau and its associated pathophysiology from those that are non-specific measures of neurodegeneration or neuronal injury.[14–19] This refinement enables identification of tauopathies and neurodegeneration or neuronal injuries that are associated, and not associated, with each other, leading to a more precise understanding of the biological underpinnings of brain ageing. To this end, an international group has proposed a new descriptive classification construct[20] for biomarkers used in Alzheimer's disease and cognitive ageing research. The construct is called ATN[20] and is based on grouping biomarkers into three categories: fibrillary β-amyloid

deposition or associated pathophysiology (A);[19] paired helical filament tau or associated pathophysiology (T);[14–19] and neurodegeneration or neuronal injury (N). One possible implementation of ATN is to dichotomise each biomarker category as either normal (–) or abnormal (+), which results in eight different biomarker group combinations. We use the terms normal and abnormal in this paper, rather than negative and positive as in previous reports, as we can know whether a scan is normal or abnormal but not whether a normal appearing scan is truly negative (ie, that no plaques or tangles would be present if the person were to come to autopsy).

The goal of this study was to apply the ATN categorisation to cognitively unimpaired individuals aged 50 years and older in the population-based Mayo Clinic Study of Aging (MCSA)[21] to estimate the age-specific and sex-specific prevalence of each ATN group, and to describe the clinical and demographic characteristics of individuals in each group. We used amyloid PET to define A, tau PET to define T, and cortical thickness to define N.

Methods

Study design and participants

We did a cross-sectional study of participants enrolled in the MCSA, a population-based study of cognitive ageing among residents of Olmsted County (MN, USA).[21] The Rochester Epidemiology Project[22] medical records linkage system was used to enumerate all Olmsted County residents aged 50–89 years. Potential participants were randomly selected from this enumeration, stratified by age and sex, with equal numbers of men and women in each age category. The random selection was achieved by randomly ordering the population enumeration in lists based on age-stratification and sex-stratification; potential participants were selected from those ordered lists by taking the first individual on each list who had not already been selected until the target enrolment in each strata was achieved. All individuals without a medical contraindication were invited to participate in imaging studies. Since 2004, the MCSA has enrolled individuals without dementia aged 70–89 years; in 2012, the study started to enrol individuals aged 50 years and older.[7,8,21] Before May 28, 2015, imaging included amyloid PET, ^{18}F-FDG PET, and MRI. From May 28, 2015, individuals who participated in imaging underwent each of amyloid PET, tau PET, and MRI.[23]

Individuals from the MCSA were included in our cross-sectional study if they were judged clinically to have no cognitive impairment and had undergone amyloid PET, tau PET (in a subset), and MRI between Oct 11, 2006, and Oct 5, 2016. We analysed data from the first visit with amyloid PET, tau PET, and MRI, or the most recent amyloid PET and MRI visit if no tau PET was available, to estimate the age-specific and sex-specific prevalence of each ATN group and to describe the clinical and demographic characteristics of the eight ATN biomarker groups.

The MCSA and related studies were approved by the Mayo Clinic and Olmsted Medical Center institutional review boards and written informed consent was obtained from all participants before they joined the study.

Procedures

Amyloid PET imaging was done with ^{11}C-Pittsburgh Compound B, synthesised on site with precursor purchased from ABX Biochemical Compounds (Radeberg, Germany). Tau PET was done with AV1451, synthesised on site with precursor supplied by Avid Radiopharmaceuticals (Philadelphia, PA, USA).[17] Late-uptake amyloid PET images were acquired 40–60 min, and tau PET 80–100 min, after injection. Methods of amyloid PET data analysis have been described previously.[7,23] We expressed amyloid PET values both as standard uptake value ratio (SUVR) units and as centiloid units.[24] A tau PET composite reporter region of interest (ROI) was formed from a voxel-number-weighted average of the median uptake in the entorhinal, amygdala, parahippocampal, fusiform, inferior temporal, and middle temporal ROIs normalised to the cerebellar crus grey median.[23] PET data were not partial-volume corrected.

MRI was done with one of three 3-Tesla systems from the same vendor (General Electric, Waukesha, WI, USA). The primary MRI measure was a FreeSurfer (version 5.3)-derived temporal lobe cortical thickness composite reporter ROI of the entorhinal, inferior temporal, middle temporal, and fusiform ROIs.[23] These were consistently among the top-performing ROIs across our previous ROI selection studies discriminating between A– cognitively unimpaired and A+ cognitively impaired individuals.[25,26] As an alternative measure of neurodegeneration we used the sum of right and left hippocampal volumes from FreeSurfer, adjusted for total intracranial volume as described previously.[27] The MRI acquisition also included a fluid-attenuated inversion recovery (FLAIR) sequence from which white matter hyperintensity volume was measured using an algorithm that we had developed previously.[28]

We had previously examined several different methods for selecting cutoff points to define abnormality with amyloid PET, tau PET, and MRI thickness.[23] The optimum amyloid PET cutoff point of SUVR 1·42 (centiloid 19) was based on the threshold value beyond which the rate of change in amyloid PET reliably increases. We determined cutoff points for tau PET and MRI thickness by maximising the accuracy (ie, maximising sensitivity plus specificity) in discriminating between A+ individuals with mild cognitive impairment or dementia versus MCSA cognitively unimpaired individuals aged 30–49 years. Based on this method, the cutoff point for tau PET was 1·23 SUVR and for MRI cortical thickness was 2·67 mm.

Each participant was classified into one of the eight ATN states using the predefined cutoff points, and we determined age-specific and sex-specific prevalences of

the eight ATN biomarker groups. As a secondary analysis, abnormal N was defined as hippocampal volume adjusted for total intracranial volume of less than –1·15 mL (HVa). This cutoff point was derived in the same manner and using the same samples described previously.[23]

Statistical analysis

The MCSA sampled similar numbers of individuals within 5-year age and sex strata from age 50 to 90 years. As a result, individuals in the older age strata were over-represented relative to the population. Therefore, to summarise the overall clinical and demographic characteristics of the eight ATN groups (appendix), we weighted our sample to reflect the actual age and sex distributions of the Olmsted County cognitively unimpaired population. Census bureau estimates for 2010 along with mild cognitive impairment and dementia-prevalence estimates from the MCSA were used to create the weights, and the survey package in R was used to correct SEs to account for strata weights (appendix).

See Online for appendix

We estimated the prevalence of each of the eight ATN groups by partitioning the full eight-group model into two components: a multinomial model with the four-level AN group as the response and age and sex as covariates (n=1548); and a logistic model with T+ as the response and AN, age, and sex as covariates (n=435). In this framework, the individuals without tau imaging can stabilise the overall prevalence estimates of the ATN groups by contributing information to the first part of the model. Inference from the model was based on posterior simulations using the maximum likelihood estimate and the variance covariance matrix. These simulations allowed us to obtain point estimates and 95% CIs for functions of the model variables such as prevalence estimates, differences in prevalence estimates, and the age at which a prevalence curve peaks (appendix).

Role of the funding source

The funders of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report. All authors had full access to all the data in the study. The corresponding author had final responsibility for the decision to submit for publication.

	A–T–N– n=165 (38%)	A–T+N– n=35 (8%)	A–T–N+ n=63 (14%)	A–T+N+ n=19 (4%)	A+T–N– n=44 (10%)	A+T+N– n=25 (6%)	A+T–N+ n=35 (8%)	A+T+N+ n=49 (11%)
Age (years)								
Median (IQR)	65 (58 to 69)	68 (62 to 78)	75 (67 to 81)	79 (73 to 82)	71 (66 to 78)	77 (70 to 82)	82 (77 to 85)	82 (76 to 87)
Range	51 to 84	53 to 90	53 to 90	63 to 95	53 to 88	65 to 94	66 to 91	64 to 94
Sex								
Male	83 (50%)	19 (54%)	45 (71%)	9 (47%)	16 (36%)	15 (60%)	22 (63%)	27 (55%)
Female	82 (50%)	16 (46%)	18 (29%)	10 (53%)	28 (64%)	10 (40%)	13 (37%)	22 (45%)
Education (years)	16 (13 to 16)	16 (14 to 17)	15 (13 to 16)	16 (13 to 16)	14 (13 to 17)	14 (14 to 17)	14 (12 to 16)	14 (12 to 16)
APOE ε4 carriers	30 (19%)	5 (15%)	13 (22%)	1 (5%)	20 (49%)	13 (52%)	14 (41%)	16 (33%)
WMH volume (mL)	5·3 (3·4 to 9·7)	6·8 (3·7 to 10·4)	11·9 (5·6 to 17·3)	15·0 (8·2 to 20·7)	9·6 (5·2 to 15·5)	8·9 (6·4 to 16·0)	19·1 (9·7 to 33·1)	18·9 (11·1 to 31·7)
Cognitive *Z* scores								
Memory	0·5 (–0·2 to 1·0)	–0·1 (–0·5 to 0·7)	–0·2 (–0·8 to 0·6)	0·2 (–0·8 to 0·9)	0·1 (–0·5 to 0·5)	0·1 (–0·8 to 0·8)	–0·6 (–1·1 to –0·1)	–0·5 (–1·1 to 0·3)
Attention	0·4 (–0·1 to 0·9)	0·6 (0·0 to 0·9)	–0·2 (–0·8 to 0·3)	0·2 (–0·2 to 0·8)	0·1 (–0·5 to 0·6)	–0·2 (–0·7 to 0·3)	–0·3 (–0·8 to 0·2)	–0·5 (–1·4 to 0·0)
Language	0·3 (–0·3 to 0·9)	0·5 (–0·2 to 1·1)	–0·2 (–0·7 to 0·3)	0·0 (–0·4 to 0·4)	0·2 (–0·2 to 0·8)	0·0 (–0·6 to 0·4)	–0·6 (–1·0 to 0·0)	–0·3 (–0·8 to 0·1)
Visuospatial	0·3 (–0·3 to 0·9)	0·3 (–0·2 to 0·8)	–0·2 (–0·7 to 0·4)	0·1 (–0·4 to 0·5)	0·1 (–0·8 to 0·5)	0·2 (–0·6 to 0·7)	–0·4 (–0·8 to 0·0)	–0·1 (–1·3 to 0·4)
Amyloid PET								
SUVR	1·31 (1·26 to 1·35)	1·33 (1·30 to 1·37)	1·33 (1·28 to 1·37)	1·35 (1·31 to 1·37)	1·57 (1·47 to 1·77)	1·62 (1·55 to 2·10)	1·58 (1·50 to 1·77)	2·22 (1·54 to 2·44)
Centiloid	9 (5 to 12)	11 (8 to 14)	11 (7 to 14)	13 (9 to 14)	31 (23 to 48)	35 (29 to 76)	32 (25 to 48)	86 (28 to 105)
Tau PET (SUVR)	1·15 (1·11 to 1·19)	1·28 (1·25 to 1·30)	1·17 (1·10 to 1·20)	1·28 (1·25 to 1·30)	1·16 (1·13 to 1·20)	1·27 (1·25 to 1·34)	1·16 (1·12 to 1·20)	1·30 (1·26 to 1·36)
Cortical thickness (mm)	2·79 (2·73 to 2·85)	2·78 (2·75 to 2·87)	2·59 (2·53 to 2·62)	2·59 (2·52 to 2·62)	2·76 (2·72 to 2·82)	2·76 (2·72 to 2·78)	2·59 (2·47 to 2·63)	2·56 (2·51 to 2·62)

Data are median (IQR) or number (%), unless stated otherwise. ATN=amyloid, tau, and neurodegeneration or neuronal injury. A–=amyloid normal using amyloid PET. A+=amyloid abnormal using amyloid PET. T–=tau normal using tau PET. T+=tau abnormal using tau PET. N–=neurodegeneration or neuronal injury normal using cortical thickness. N+=neurodegeneration or neuronal injury abnormal using cortical thickness. WMH=white matter hyperintensities. SUVR=standard uptake value ratio.

Table 1: **Characteristics of 435 participants by ATN biomarker classification**

Results

Table 1 shows unweighted data in our ATN sample (n=435). Summaries by ATN group weighted to the cognitively unimpaired Olmsted County population by age and sex are in figure 1. Age differed among ATN groups (p<0·0001) with individuals with worse biomarker profiles tending to be older (table 1, figure 1). The group with the greatest estimated proportion of men was A–T–N+ (57%, 95% CI 37–77) and the group with the greatest proportion of women was A+T–N– (78%, 64–93); however, overall the sex distribution was not different among the ATN groups (p=0·21). *APOE* ε4 varied by ATN group (p=0·04) and was roughly twice as frequent among A+ individuals compared with A– individuals. White matter hyperintensity volume differed between ATN groups (figure 1, p<0·0001) even after adjustment for age (p=0·01; appendix), and was higher in N+ than N– groups (p=0·05; not shown), although the magnitude of the differences was small. There were small, but significant, differences in cognition by group for all domains (figure 1, p<0·0001) even after adjustment for age (appendix, p≤0·03).

The appendix shows demographic features of the full sample of 1548 cognitively unimpaired MCSA individuals with amyloid PET and MRI (but not necessarily tau PET) who were used to constrain or stabilise ATN-prevalence estimates among the subset of 435 who had amyloid PET, MRI, and tau PET.

For both men and women, A–T–N– prevalence declined from age 50 years onwards, while that of A–T+N+ increased gradually with age starting at 60 years, and that of A+T+N+ increased more markedly with age beginning in the late 60s (figure 2). The remaining ATN groups reached individual peaks in prevalence. Comparisons of

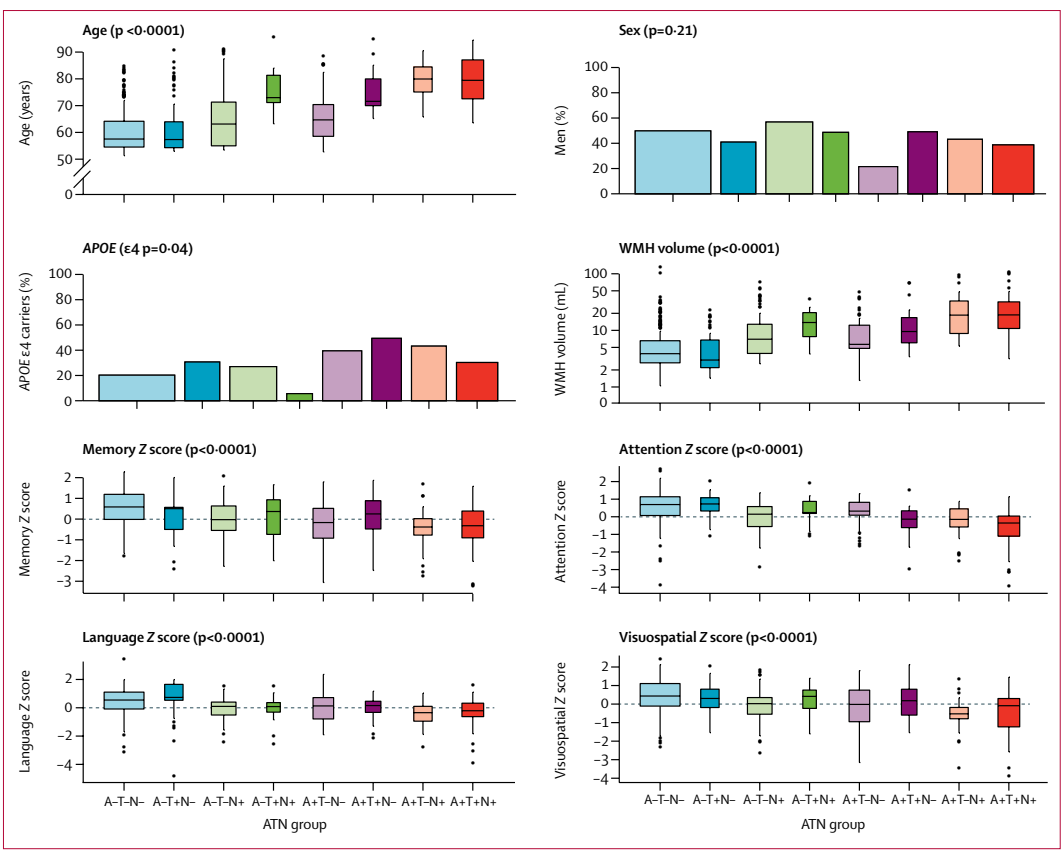

***Figure 1*: ATN group characteristics**

Box plots of continuous variables and bar charts summarising percentages of categorical variables from table 1 by ATN biomarker group. The box plots and estimated percentages reflect weighting of the sample to match the age distribution and sex distribution of the sample population (Olmsted County [MN, USA] residents who were clinically normal). Box and bar widths reflect sample sizes. p values test for any difference in each variable among the eight groups. The box plot whiskers extend to the lowest and highest data points within 1·5 times the IQR from the lower and upper quartiles. The dots represent individual points that fall outside this range. ATN=amyloid, tau, and neurodegeneration or neuronal injury. A–=amyloid normal using amyloid PET. A+=amyloid abnormal using amyloid PET. T–=tau normal using tau PET. T+=tau abnormal using tau PET. N–=neurodegeneration or neuronal injury normal using cortical thickness. N+=neurodegeneration or neuronal injury abnormal using cortical thickness. WMH=white matter hyperintensities.

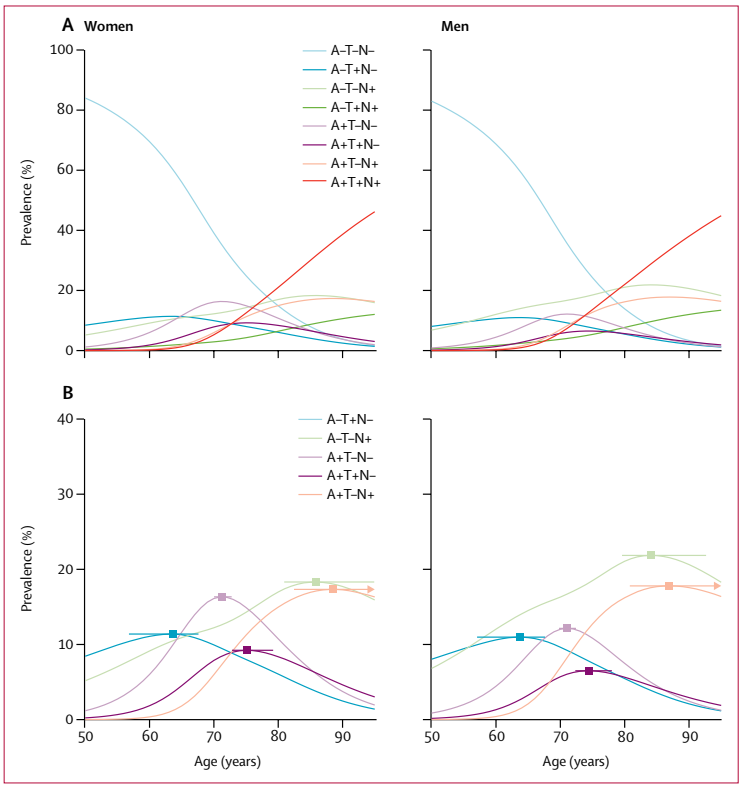

***Figure 2*: Estimated prevalence of the ATN biomarker groups by age and sex**
Estimated prevalence curves by age and sex for all ATN groups (A) and the same curves (except for A–T–N–, A–T+N+, and A+T+N+) on an enlarged scale with the estimated peak for each curve shown with a square and 95% CI (B). Arrows represent CIs that extended past the x-axis limits of the figure (ie, where the upper limit was 100). ATN=amyloid, tau, and neurodegeneration or neuronal injury. A–=amyloid normal using amyloid PET. A+=amyloid abnormal using amyloid PET. T–=tau normal using tau PET. T+=tau abnormal using tau PET. N–=neurodegeneration or neuronal injury normal using cortical thickness. N+=neurodegeneration or neuronal injury abnormal using cortical thickness.

	Women, peak age (years)	Men, peak age (years)	Differences in peak age (years)
A–T+N–	64 (57 to 68)	64 (57 to 68)	0 (–1 to 2)
A+T–N–	71 (70 to 73)	71 (70 to 72)	0 (–1 to 0)
A+T+N–	75 (73 to 79)	74 (72 to 78)	–1 (–2 to 0)
A–T–N+	86 (81 to 95)	84 (80 to 93)	–2 (–4 to 0)
A+T–N+	88 (82 to 100)	87 (81 to 100)	–2 (–4 to 1)

Data are peak age (95% CI), or differences in peaks by sex. ATN=amyloid, tau, and neurodegeneration or neuronal injury. A–=amyloid normal using amyloid PET. A+=amyloid abnormal using amyloid PET. T–=tau normal using tau PET. T+=tau abnormal using tau PET. N–=neurodegeneration or neuronal injury normal using cortical thickness. N+=neurodegeneration or neuronal injury abnormal using cortical thickness. Peak ages are not shown for A–T–N– because the prevalence declined over the entire age range, or for A–T+N+ or A+T+N+ because the prevalence of these groups increased over the entire age range.

***Table 2*: Age at which the percentage of each ATN prevalence curve reaches its peak for women and men**

the curves for men versus women (appendix) revealed a slightly greater prevalence of A–T–N+ in men from age 65 to 75 years but no other clear sex differences.

We averaged the sex-specific prevalence estimates to make direct age-specific prevalence comparisons between ATN groups (appendix). The dominant trends were that A–T–N– was the most prevalent group from age 50 years to the late 70s, and that A+T+N+ was the most prevalent group from age early 80s onwards.

The ages at which the prevalence curves peaked differed substantially among ATN groups but were similar for men and women within each group (table 2, figure 2). A–T+N– was the first group to peak (age 64 years), followed by A+T–N– and A+T+N– (ages 71 and 74–75 years, respectively). The N– groups all peaked by age 75 years or younger, whereas the N+ groups did so at or above age 84 years. Differences in peak age between some ATN groups were substantial (appendix), particularly between N– and N+ groups. For example, the A+T–N+ and A–T–N+ groups peaked 25 years (95% CI 15–42) and 22 years (14–34) later than the A–T+N– group.

Figure 3 illustrates that abnormalities in A, T, and N mostly did not overlap at young ages. At older ages, the presence of more than one abnormal biomarker was common and there was substantial discordance among the three. Tau and neurodegeneration were discordant in 86% of individuals categorised as NIA-AA preclinical Alzheimer's disease stage 2 or 3 (ie, individuals classified as A+T+N– or A+T–N+ as a proportion of those classified as A+T+N–, A+T–N+, or A+T+N+) at age 65 years and in 51% at age 80 years (figure 3). Tau PET and neurodegeneration biomarkers were also discordant in most individuals categorised as SNAP (ie, individuals classified as A–T+N– or A–T–N+ as a proportion of those classified as A–T+N–, A–T–N+, or A–T+N+; 92% at age 65 years and 78% at age 80 years; figure 3). ATN prevalence by age was also calculated using HVa instead of cortical thickness as the N measure (appendix). Although agreement between measures of HVa and thickness was moderate (κ=0·45), overall the ATN prevalence trends by age were similar when either measure was used. One difference was a higher prevalence of N+ in men than in women when using HVa, which was evident when comparing the A–T–N+ curves (figure 2, appendix).

Discussion

Our main findings were that A–T–N– prevalence declined from age 50 years onwards whereas the prevalence of A–T+N+ and A+T+N+ increased continuously with age for both men and women. A–T–N– was the most prevalent group from age 50 years to the late 70s. From age late 70s onwards, A+T+N+ was the most prevalent group. The other N– groups (A–T+N–, A+T–N–, and A+T+N–) all reached peak prevalence by age 75 years or younger whereas the other N+ groups (A–T–N+ and A+T–N+) reached a peak prevalence at or above age 84 years.

Cross-sectional prevalence curves are the first step in understanding the complex and interdependent evolution of amyloid, tau, and neurodegeneration in ageing individuals. Because our sample came from a geographically stable population, secular changes are likely to be minimised, and thus we interpreted differences in ATN prevalence curves across the 50–90 years age range as being largely due to transitions between biomarker groups as people age. The declining prevalence of A–T–N– with age is logical, since individuals can only transition out of A–T–N–, while the increasing prevalence of A+T+N+ with age makes sense because this is an absorbing biomarker state—ie, people can transition out of all states except A+T+N+. The increasing prevalence of A–T+N+ might reflect an absorbing state for those on a non-Alzheimer's disease pathway.

For the other five ATN groups, the prevalence increased to a peak with age, then declined. The age at which the prevalence curves peaked differed, but peak ages can be grouped into two clusters. The N– groups with evidence of either abnormal amyloid or tau deposition all peaked by age 75 years or younger, whereas the N+ groups peaked at or above age 84 years. From age 75–85 years, the prevalence of the three N– groups fell whereas the prevalence of the two N+ groups rose. This finding is consistent with the idea that neurodegeneration or neuronal injury is a downstream consequence of antecedent proteinopathies. The fact that A–T–N+ was more frequent than some N– groups in middle age is consistent with the idea that this group is on a separate, non-Alzheimer's disease trajectory where neurodegeneration is not driven by Alzheimer's disease proteinopathy.

Overall the effect of sex on the prevalence of all ATN groups was small when using cortical thickness as a measure, but more pronounced when using HVa. *APOE* ε4 was more frequent among the A+ than the A– groups. Among individuals who were A–, we noted no clear evidence of elevation in *APOE* ε4 frequency among A–T+N–, A–T–N+, or A–T+N+ relative to A–T–N–. Similarly, among individuals who were A+, we showed no evidence of elevation in *APOE* ε4 frequency among A+T+N–, A+T–N+, or A+T+N+ relative to A+T–N–. One interpretation of this finding is that the primary effect of *APOE* ε4 is to increase amyloidosis, not to enhance tau deposition, neurodegeneration, or both through non-amyloid-related mechanisms.

Abnormal biomarker profiles were associated with worse cognition across different domains after adjusting for age. White matter hyperintensity volume was higher in N+ than in N– groups (p=0·05). This finding supports the position that ischaemic brain injury is, among other conditions,[29] a likely contributor to N+.

SNAP was first described as A–N+ where N+ was based on ^{18}F-FDG PET and MRI findings.[7] In the 2011 NIA-AA criteria, the definition of N+ also included abnormal CSF phosphorylated and total tau.[1–4] In our data, 15% of individuals were classified as SNAP defined by MRI and amyloid PET at age 65 years, and 26% at age 80 years. Of these individuals, 13% at age 65 and 27% at age 80 also had abnormal tau PET (ie, A–T+N+). Thus, tau and neurodegeneration were concordant only in a few A–N+ (SNAP) individuals for whom N+ was defined by cortical thickness.[30,31] Mormino and colleagues[32] and Wisse and colleagues[33] have reported that tau was not elevated in SNAP relative to A–N– individuals who were classified by amyloid PET and hippocampal volume or ^{18}F-FDG PET using the NIA-AA staging plus SNAP construct. Similarly, we noted that the proportions of T+ participants

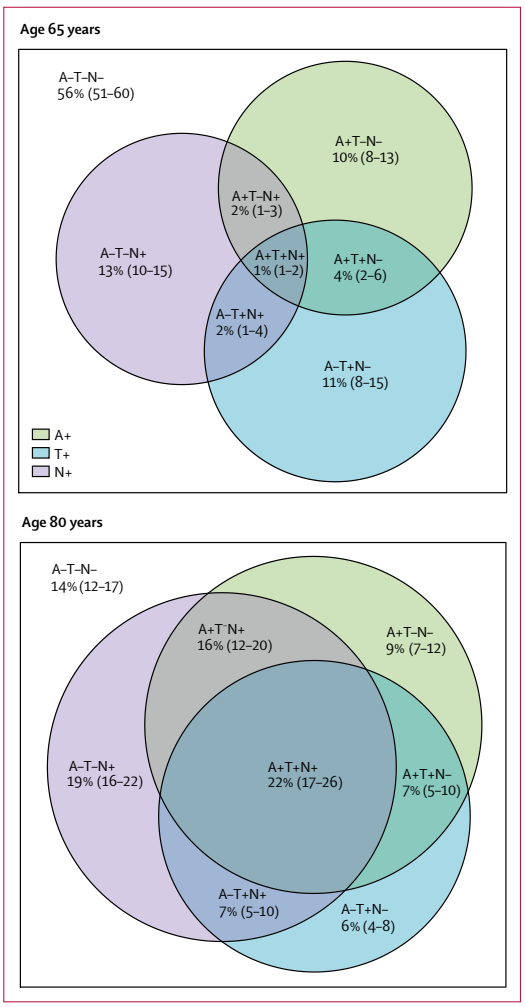

***Figure 3*: Estimated prevalence of each ATN group at ages 65 and 80 years.** Data are estimates (95% CIs) averaged over men and women. Since estimates were for a given age among clinically normal individuals, weighting to the population was not necessary. ATN=amyloid, tau, and neurodegeneration or neuronal injury. A–=amyloid normal using amyloid PET. A+=amyloid abnormal using amyloid PET. T–=tau normal using tau PET. T+=tau abnormal using tau PET. N–=neurodegeneration or neuronal injury normal using cortical thickness. N+=neurodegeneration or neuronal injury abnormal using cortical thickness.

were similar among the A–N– and A–N+ groups (16% *vs* 13% at age 65 years and 30% *vs* 27% at age 80 years). However, by classifying A, T, and N separately, we showed that tau PET is frequently abnormal in SNAP where N+ is defined by cortical thickness. Tau PET had not yet been studied in humans when SNAP was first described in 2012.[7] If the A–T+N– profile is included in the SNAP category where T+ is defined by tau PET, which we believe should be the case, then the proportion of SNAP with evidence of tauopathy is 50% at age 65 and 41% at age 80 years.

We postulate that the A–T–N+ profile corresponds to neurodegeneration due to a heterogeneous group of non-Alzheimer's disease pathologies that increase in prevalence with age including cerebrovascular disease, Lewy body disease, TDP 43, argyrophilic grains, and hippocampal sclerosis.[34] A logical assumption is that the A–T+N– profile corresponds to primary age-related tauopathy.[35] The A–T+N+ profile might correspond to a combination of primary age-related tauopathy and other non-Alzheimer's disease pathologies. However, imaging–autopsy correlation studies will be needed to confirm these hypotheses.

The four A+ profiles represent preclinical Alzheimer's disease according to the 2011 NIA-AA guidelines. Tau and neurodegeneration were discordant in 86% of individuals categorised as NIA-AA preclinical Alzheimer's disease stage 2 or 3 at age 65 years and in 51% at age 80 years. A model of Alzheimer's disease pathogenesis proposes that amyloidosis promotes increased local tau deposition and its spread, which in turn is responsible for neurodegeneration.[36] The ATN biomarker counterpart would be a sequence of A+T–N– to A+T+N– to A+T+N+. The median ages of these three groups and the ages at which the prevalence curves peak increase incrementally; this evidence lends support to the hypothesis that A+T–N– to A+T+N– to A+T+N+ is the biomarker sequence of preclinical Alzheimer's disease. However, longitudinal data will be necessary to confirm this sequence. The A+T–N+ profile, which does not fit into the sequence of preclinical Alzheimer's disease we propose, perhaps indicates individuals in whom two different types of pathology are evident by biomarkers: a non-Alzheimer's disease degenerative process resulting in N+, plus early Alzheimer's disease resulting in the A+T– profile.

For our primary analyses, we used cortical thickness rather than commonly used hippocampal volume as our measure of neurodegeneration to avoid necessitating an adjustment for head size. Brain volumes scale with head size,[37] and correcting for this is not straightforward since head size is related to sex, yet sex-specific effects on atrophy probably exist. A solution is to use cortical thickness, which does not scale closely with head size and consequently does not require an adjustment.[37] Overall, the ATN prevalence curves by age were similar when either HVa or cortical thickness was used as the N measure. These findings suggest that the prevalence of ATN groups we report should be robust to different definitions of N. However, with only moderate agreement between abnormal HVa and thickness, there might be differences in which individuals are labelled N+ by the two biomarkers. We are uncertain whether the more pronounced sex differences when using HVa as the N measure represent an artifact of head size adjustment or a true biological effect.

Our use of the ATN scheme reflected several methodological factors and decisions. Both clinical-imaging correlation[14–18] and autoradiographic[38,39] evidence point to AV1451 as a useful measure of the 3R/4R paired helical filament tau deposits that are characteristic of Alzheimer's disease and primary age-related tauopathy.[35] Binding in primary tauopathies (except those that produce 3R/4R fibrillar tau deposits) is however less certain. In our study, we used a single-reporter tau PET meta-ROI that included medial, basal, and lateral temporal lobe areas.[23] Our rationale was that tau PET uptake in these areas is consistently associated with characteristics of Alzheimer's disease such as the presence of amyloid on PET, worse cognitive performance across the clinical spectrum, and abnormal CSF phosphorylated tau.[14–18] This set of ROIs captures a broad dynamic range across the normal to pathological ageing to Alzheimer's disease dementia spectrum; it therefore seems to represent a reasonable tau PET summary measure.[23]

The ATN framework requires definition of abnormality in each biomarker. We have previously examined different methods for selecting cutoff points to define abnormality on amyloid PET, tau PET, and cortical thickness.[23] We regard plaques, tangles, and synapse loss to be pathological. Although all of these processes increase in frequency and severity with age,[34] our cutoff points were not age-adjusted. We believe that, although not age-norming the cutoff points results in a greater proportion of older individuals being labelled abnormal, the fact that an entity is frequent does not disqualify it from being pathological. Age-norming of cognitive tests is common practice, but biomarkers in other fields are typically not age-normed. For example, the cutoff points used to define diabetes or hypertension do not change with age. Loss of synapses and dendritic spines and associated cognitive or functional loss seem to be a nearly universal feature of ageing in human beings and a range of animal species.[40,41] Whether these losses should be considered pathological or not is an unresolved question.

The methods of selecting reporter meta-ROIs and cutoff points used in ATN classification centred around Alzheimer's disease. However, although temporal lobe atrophy is characteristic of Alzheimer's disease, it is not diagnostic for this condition. Many non-Alzheimer's disease disorders (eg, argyrophillic grains and hippocampal sclerosis) might produce atrophy in these brain areas. However, until specific biomarkers of the common non-Alzheimer's disease entities are developed, the only available biomarker evidence of their presence are non-specific indicators of neurodegeneration or neuronal injury.

Our study had limitations. For example, because eight possible ATN combinations exist, participant numbers in some groups were small. Dichotomising each biomarker simplifies what is an underlying continuous process. Measurement imprecision inevitably results in some classification errors, particularly for values close to cutoff points. With three different biomarker classes per individual, the likelihood of classification error is compounded compared with the use of only one biomarker. We did not examine individuals in the population who had become clinically impaired; this awaits greater enrolment of impaired individuals in the MCSA. While the most rational explanation for the changing prevalence of ATN groups with age is within-individual ATN group transitions, our data are cross-sectional. Our study raises questions for which no answers exist currently; for example, what are the longitudinal clinical or cognitive outcomes and the pathological underpinnings of these ATN groups? Answers to such questions require longitudinal clinical follow-up in many well characterised individuals with eventual autopsy correlation. To our knowledge, such data do not currently exist for individuals characterised by ATN profiles, and data addressing these issues await maturation of ours and others' research cohorts.

Contributors

CRJ Jr conceptualised the study, analysed and interpreted the data, and drafted and revised the manuscript. HJW and SDW conceptualised the study, analysed and interpreted the data, drafted and revised the manuscript, and did the statistical analysis. TMT analysed and interpreted the data, drafted and revised the manuscript, and did the statistical analysis. DSK, PV, MMMi, ROR, MMMa, and WAR drafted and revised the manuscript. VL collected the data and drafted and revised the manuscript. MLS, JLG, and RCP analysed and interpreted the data and drafted and revised the manuscript.

Declaration of interests

CRJ Jr reports grants from the US National Institutes of Health (NIH), and other finances from Eli Lilly. DSK reports personal fees from the data safety monitoring board of the DIAN study, and personal fees from the data safety monitoring board of Lundbeck Alzheimer's disease trial. VL reports personal fees from Bayer Pharmaceuticals, grants from GE Health Care, grants from Siemens Molecular Imaging, grants from AVID Radiopharmaceuticals, personal fees from Piramal Imaging, and personal fees from Merck Research. MLS reports stock or options ownership from Celgene Corporation, Inovio Pharmaceuticals, Medtronic, Parexel International Corporation, and Gilead Sciences. RCP reports grants from the NIH during the study, and personal fees from Hoffman-La Roche, Merck, Genentech, Biogen, and Eli Lilly. The other authors declare no competing interests.

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FEATURED ARTICLE

Associations among vascular risk factors, neuroimaging biomarkers, and cognition: Preliminary analyses from the Multi-Ethnic Study of Atherosclerosis (MESA)

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Abstract

Introduction: Little is known about how antecedent vascular risk factor (VRF) profiles impact late-life brain health.

Methods: We examined baseline VRFs, and cognitive testing and neuroimaging measures (β-amyloid [Aβ] PET, MRI) in a diverse longitudinal cohort (N = 159; 50% African-American, 50% White) from Wake Forest's Multi-Ethnic Study of Atherosclerosis Core.

Results: African-Americans exhibited greater baseline Cardiovascular Risk Factors, Aging, and Incidence of Dementia (CAIDE), Framingham stroke risk profile (FSRP), and atherosclerotic cardiovascular disease risk estimate (ASCVD) scores than Whites. We observed no significant racial differences in Aβ positivity, cortical thickness, or white matter hyperintensity (WMH) volume. Higher baseline VRF scores were associated with lower cortical thickness and greater WMH volume, and FSRP and CAIDE were associated with Aβ. Aβ was cross-sectionally associated with cognition, and all imaging biomarkers were associated with greater 6-year cognitive decline.

Discussion: Results suggest the convergence of multiple vascular and Alzheimer's processes underlying neurodegeneration and cognitive decline.

KEYWORDS
aging, cognition, magnetic resonance imaging, positron emission tomography, vascular risk factors

1 | BACKGROUND

Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by brain β-amyloid (Aβ) and tau pathology and cognitive decline, and culminating in dementia.[1,2] Brain positron emission tomography (PET) measures of Aβ pathology precede early cognitive symptoms of AD.[3–5] AD pathology occurs close in time to the development of cerebral small vessel disease (SVD) in the mid- to late-life transition period. Magnetic resonance imaging (MRI)[6–8] has shown that SVD impacts white matter (WM) health and brain networks including elevations in WM hyperintensities (WMH),[9,10] an MRI biomarker of SVD. Further, measures of cortical atrophy serve as a biomarker of neurodegeneration related to numerous disease pathologies, including both SVD and AD.[11]

While AD and SVD are thought to be independent processes, vascular risk factors (VRFs) are associated with both forms of disease. Growing evidence suggests that VRFs (eg, hyperlipidemia, systolic blood pressure [BP], type 2 diabetes, arterial stiffness) relate to not only SVD-linked imaging markers, but also AD-linked pathological measures such as atrophy and Aβ accumulation.[12–14] In addition, while late-life VRFs are not associated with dementia and its related pathology, midlife VRFs tend to be associated with pathology (particularly imaging biomarkers) and cognition; one study found elevated midlife VRFs were significantly associated with elevated Aβ on PET, consistent with a role of vascular disease in the development of AD.[15]

VRFs are also being used to stratify risk in dementia prevention trials. The FINGER[16] study, which recruited older individuals with a high Cardiovascular Risk Factors, Aging, and Incidence of Dementia (CAIDE) risk score,[17] found a multidomain intervention targeting VRFs (diet, physical activity, VRF management) helped to protect against cognitive decline. In the small FINGER imaging substudy, CAIDE was associated with more deep WMH and lower cortical thickness, but not with Aβ or periventricular WMH.[18] These studies often lack sufficient diversity to explore relationships between VRFs, neuropathology, and cognition in older adults from different racial groups.

The Multi-Ethnic Study of Atherosclerosis (MESA) is a unique diverse study with over 20 years of extensive longitudinal vascular phenotyping, as well as cognitive testing 10 and 15 years later.[19,20] At the Wake Forest University (WFU) site, a diverse (White and African-American [AA]) cohort additionally underwent brain MRI and Aβ PET. The examination of imaging biomarkers in this deeply phenotyped older adult sample is important to understanding the role of VRFs in neuropathology and cognitive decline.

In this study, we examined relationships among (1) various composite VRF measures, (2) imaging biomarkers of SVD and AD-related pathology, and (3) cognitive performance and change in cognition, in a diverse sample of community-dwelling older adults. Multiple commonly used VRF scores were evaluated in order to better understand contributions of vascular risk to neuropathology and cognitive decline. We hypothesized elevated antecedent VRF scores (assessed by clinical vascular composites 10-16 years prior to neuroimaging and cognitive testing) would be differentially associated with imaging biomarkers of Aβ (global Pittsburgh compound B [PiB] PET[3,21]), SVD (global WMH volume[22]), and neurodegeneration (temporal lobe MRI cortical thickness[11]), and these baseline VRF scores and imaging biomarkers would predict cognitive performance. We additionally examined whether there would be differences by race in the relationship between VRF scores and brain imaging biomarkers.

2 | METHODS

2.1 | Participants

Participants were recruited from the parent MESA cohort study into the WFU Alzheimer's Disease Research Center affiliated MESA Core study. MESA Core participants (N = 159; 49.7% AA and 50.3% White) were included in this analysis. As previously described,[19,20] MESA participants were free from clinical cardiovascular disease (including stroke) at baseline (2000-2002, when participants were 55.8 ± 6.7 years old) and underwent VRF assessment over the course of six exams and annual follow-up calls; baseline VRF values were analyzed in this study. Participants underwent cognitive testing with the Cognitive Abilities Screening Instrument (CASI)[23] at two timepoints, first in 2010-12 (MESA Exam 5) and again in 2016-2018 (Exam 6). At the WFU site, participants also received more detailed cognitive testing, brain MRI and PET as part of MESA Core (2016-2018). DNA was analyzed for *APOE* genotypes as previously described;[24] *APOE*-ε4 carriage was defined as the presence of one or more ε4 allele(s). Participants self-reported their sex, racial group, and years of education. All participants were free from clinical stroke. The research protocol was approved by the local Institutional Review Board, and informed consent was obtained for all participants; all research was carried out in accordance with the Declaration of Helsinki.

2.2 | Vascular risk factor composite scores

Multiple composite baseline (2000-2002) mid- to late-life clinical VRF scores were evaluated: CAIDE,[17] Framingham stroke risk profile (FSRP) score,[25] and the atherosclerotic cardiovascular disease risk estimate from the pooled cohort equation (ASCVD).[26] CAIDE is a composite of traditional VRFs (eg, systolic BP, cholesterol) and AD risk factors (eg, education, *APOE*-ε4). It is a useful predictor of dementia

risk, with a higher score indicating greater risk.[17] FSRP estimates the risk of stroke over 10 years and includes a combination of traditional VRFs with prevalent cardiovascular disease (CVD) and atrial fibrillation. FSRP is associated with lower brain volume, higher WMH, and cognitive decrements.[27–29] ASCVD is a predictor of 10-year risk of a first ASCVD event such as nonfatal myocardial infarction or coronary heart disease. Both FSRP and ASCVD are represented as a percent risk with higher percentages indicating increased risk. Components of each composite VRF score are summarized in Supplementary Table 1 (in the Supporting Information). All VRF scores include age, sex, and systolic BP; ASCVD and FSRP share components (type 2 diabetes, use of antihypertensive medications, current smoking status), as do ASCVD and CAIDE (total cholesterol).

2.3 | Cognitive testing and adjudication

At the MESA Exam 5 (2010-2012), participants had a brief cognitive examination using the CASI.[23] This testing was repeated at MESA Exam 6 (2016-2018). Cross-sectional cognitive performance (Exam 5, Exam 6) as well as longitudinal cognitive change (Exam 5 CASI score subtracted from Exam 6 CASI) were calculated and entered into the analyses. The CASI is a measure of global cognitive function developed for cross-cultural use; however, known differences exist in CASI scores between racial/ethnic groups.[30,31] CASI scores were missing or deemed invalid by the test administrator for three AA participants at Exam 5 and 1 White participant at Exam 6; we did not evaluate cognitive performance in these four participants.

Also at Exam 6, MESA Core participants were administered the UDSv3 cognitive assessment protocol.[32] The National Alzheimer's Coordinating Centers UDSv3 cognitive assessment protocol includes detailed cognitive testing; information about family history of AD, medications, and health history; clinician-assessed medical conditions and judgment of symptoms; and a neurological examination. All of these data were used in the adjudication process. The consensus panel consisted of neuropsychologists, geriatricians, neurologists, and other aging experts. Consensus adjudicated cognitive status, according to published criteria,[33,34] included cognitively normal, mild cognitive impairment (MCI), and dementia subtypes (including AD).

2.4 | MRI acquisition and processing

Brain MRI data were acquired for all participants at WFU on a 3T Siemens Skyra scanner using a high-resolution 32-channel head coil. MRI sequences included T1-weighted 3D volumetric MPRAGE (to quantify cortical thickness) and T2-weighted FLAIR (to quantify WMH). Sequence details are provided in the supplementary information S1. Cortical thickness was calculated on T1 MRI using FreeSurfer v5.3 (https://surfer.nmr.mgh.harvard.edu). We examined brain structure using a temporal lobe region of interest; this was calculated by averaging surface area-weighted cortical thickness of bilateral entorhinal, inferior/middle temporal, and fusiform regions.[11] This cortical thickness measure has been shown to be a useful measure of neurodegeneration in regions characteristic of AD. Lesions were segmented by the lesion growth algorithm (LGA)[35] implemented in the LST toolbox v2.0.15 (www.statistical-modelling.de/lst.html), running in Matlab SPM12 (www.fil.ion.ucl.ac.uk/spm) using FLAIR images with T1 images as reference. WMH masks were manually edited by trained observers. Total WMH lesion volume was divided by FreeSurfer total intracranial volume (to correct for head size) and log-normalized to generate a global measure of WMH volume, which served as an SVD biomarker.

RESEARCH IN CONTEXT

1. Systematic review: The authors reviewed the literature using PubMed and meeting abstracts and presentations. It is poorly understood how antecedent vascular risk factor (VRF) profiles translate into late-life brain health, particularly among diverse older adults. Several recent studies have begun to investigate this question, using imaging biomarkers of disease and cognitive testing; these relevant citations are appropriately cited.
2. Interpretation: Our findings suggest that there are complex, long-acting interactions between multiple vascular and AD processes on late-life neurodegeneration, neuropathology, and cognitive decline.
3. Future directions: Future studies that incorporate multimodal variables (VRFs, imaging biomarkers, and cognitive performance) from mid- to late-life in diverse samples with longitudinal study designs are needed to clarify the complex and long-acting impacts of vascular and AD pathology on late-life brain and cognitive decline, and how they differ among racial groups.

2.5 | PET acquisition and processing

[^{11}C]PiB[36] was used for assessing fibrillar Aβ brain deposition on PET. Following a computed tomography (CT) scan for attenuation correction, participants were injected with approximately 370 MBq [^{11}C]PiB and scanned from 40-70 minutes (6 × 5-min frames) post-injection in the WFU PET research center on a 64-slice GE Discovery MI DR PET/CT scanner. Each participant's CT image was coregistered to their structural MRI, and PET frames were coregistered to MRI space using the affine matrix from the CT-MRI coregistration. A voxelwise 40-70 minutes standardized uptake volume ratio (SUVR) image was then generated. Global brain PiB uptake was calculated as PiB SUVR (40-70 minutes, cerebellar grey reference) averaged from a cortical region of interest sensitive to early AD, using FreeSurfer-segmented regions.[3,37] This global PiB SUVR measure served as a biomarker of Aβ burden. Participants were classified using a previously defined threshold (≥1.21 PiB SUVR[21]) to define Aβ- and Aβ+ groups.

2.6 | Statistical analysis

We tested differences between racial groups and Aβ positivity groups using *t* tests and chi-square tests; racial group differences in Aβ were also assessed using multivariable linear regression with covariates of age, sex, education, *APOE*-ε4, WMH volume, and intracranial volume, in line with previous studies.[38] We assessed bivariate associations between VRF scores and imaging variables using the Pearson correlation. This analysis focused on three types of brain imaging: temporal lobe cortical thickness, WMH volume, and brain Aβ burden (methods described above). We developed multivariable general linear models assessing relationships of (1) VRF scores with brain imaging variables, (2) VRF scores with cognition (CASI performance), and (3) brain imaging variables with cognition. Results from general linear models were reported as *t* values and *P* values. Linear models were adjusted for a basic set of covariates: age, sex, race, education, and *APOE*-ε4. Covariates were not included in models when they were included in VRF scores (Supplementary Table 1). Because we were interested in differences by racial groups in VRF scores and their impacts on brain health and cognition, we examined interactions by race. It is important to note that in this analysis, we interpret race as a social, and not biological, construct. We additionally examined interactions by baseline median age (54 years) to assess potential effect modifications by age, as well as interactions by cognitive impairment status (comparing cognitively normal vs MCI and dementia combined). Participants with VRF scores ≥4 standard deviations above the sample mean were removed from analyses. One participant was removed from analyses that included FSRP; results were similar when this participant was included in the analyses.

3 | RESULTS

Demographics by Aβ status for 159 participants (102 Aβ-, 57 Aβ+) in the present study are presented in Table 1. Aβ+ participants (35.8%) were more likely to be older, *APOE*-ε4 carriers, and have higher WMH volume and lower temporal lobe cortical thickness. Further, the Aβ+ group exhibited lower Exam 6 CASI scores and significantly greater decline in CASI from Exams 5 to 6; there was no difference in the Exam 5 CASI scores. A significantly greater proportion of Aβ+ participants had cognitive impairment ($P < .001$), and higher CAIDE ($P < .001$) and FSRP scores ($P = .004$).

When examining self-reported racial group membership (Table 2; 80 White, 79 AA), we found an expected significant difference in ASCVD score (race is a component of the ASCVD score), and a marginal difference in CAIDE ($P = .051$), such that AA participants had higher mid- to late-life VRF scores. Systolic and diastolic blood pressure (SBP and DBP) were significantly elevated in the AA compared to the White group. AA participants were more likely to be male ($P = .045$) and had lower overall PiB SUVR ($P = .029$), yet notably there were no racial differences in Aβ positivity ($P = .272$, Table 2) even after controlling for other covariates (Supplementary Table 2). We found no significant group differences by race in other demographic or imaging measures.

In bivariate analysis, higher baseline FSRP and CAIDE scores were significantly associated with global brain Aβ (Figure 1A). We observed that higher baseline VRF scores were significantly associated with both greater WMH volumes (Figure 1B) and reduced temporal lobe cortical thickness (Figure 1C).

In multivariable models of VRF scores as predictors of imaging variables (Table 3), we found that elevated ASCVD, FSRP, and CAIDE significantly predicted lower temporal lobe cortical thickness and higher WMH volumes, independent of covariates. CAIDE and FSRP were significantly associated with brain Aβ burden. There were no interactions with baseline age or cognitive status and VRF scores with neuroimaging biomarkers (data not shown). There were significant interactions by race with respect to cortical thickness, such that associations with ASCVD and FSRP were stronger in White participants (Supplementary Table 3).

When examining relations among VRF scores and CASI (Table 4), we found no significant associations with Exam 5 CASI scores. Higher ASCVD, FSRP, and CAIDE scores were associated with worse Exam 6 CASI performance. The association of CAIDE and Exam 6 CASI was driven primarily by the AA group (Supplementary Table 4). Elevated FSRP and CAIDE were related to greater CASI decline. There were no interactions with baseline age (data not shown). We observed an interaction by cognitive status in the ASCVD-Exam 6 CASI association; the association was strongest in cognitively normal participants (data not shown).

We finally examined multivariable models of imaging variables as predictors of CASI (Table 5). All imaging markers were significantly associated with CASI decline over 6 years. Measures of WMH and cortical thickness were not significantly associated with Exam 5 or Exam 6 CASI performance. While global PiB SUVR was not associated with the Exam 5 CASI, greater brain Aβ burden was significantly associated with lower Exam 6 CASI performance. We observed an interaction by cognitive status for associations of PiB SUVR with CASI Exam 6 and CASI Change, such that associations were strongest in cognitively impaired participants (data not shown).

4 | DISCUSSION

In this diverse cohort, we assessed relationships among baseline VRF scores, brain imaging biomarkers of SVD and AD, and cognitive performance in a diverse older adult sample. The MESA cohort enabled the study of contributions of antecedent mid- to late-life VRFs to subsequent neurodegeneration, neuropathology, and cognitive decline; additionally, MESA allowed us to assess unique associations of each composite VRF measure with late-life imaging biomarkers, and to explore whether VRFs differentially predict Aβ deposition, WMH, and cortical thickness. It additionally enabled the study of differences in specific VRF pathways and imaging markers among a diverse group of individuals. Multiple VRF scores were evaluated (CAIDE,[17] FSRP,[25] and ASCVD[26] scores) in order to better understand the contributions of vascular risk to neuropathology and cognitive decline in the context of other studies that have investigated these commonly-used VRFs.

TABLE 1 Participant measures by Aβ positivity

	Aβ-	Aβ+	*P*
n (%)	102 (64.1)	57 (35.8)	
Age, Exam 1, M (SD)	54.3 (6.3)	58.4 (6.5)	<.001*
Age, Exam 6, M (SD)	70.1 (6.2)	74.5 (6.5)	<.001*
Sex (M/F)	47/55	24/33	.629
Race (White/AA)	48/54	32/25	.272
Years of education, M (SD)	15.0 (2.6)	15.1 (2.9)	.857
[a]Cognitive status, n (%)			<.001*
Normal	79 (78.2)	27 (48.2)	
MCI	19 (18.8)	23 (41.1)	
Dementia	1 (1.0)	5 (8.9)	
APOE-ε4, (+/-/missing)	26/75/1	27/25/5	<.001*
% ASCVD, M (SD)	6.59 (5.7)	8.01 (6.4)	.152
[b]% FSRP, M (SD)	1.69 (1.6)	2.69 (2.7)	.004*
CAIDE, M (SD)	6.48 (2.2)	7.65 (2.2)	.002*
SBP, M (SD)	127.0 (21.7)	126.9 (18.6)	.965
DBP, M (SD)	73.9 (10.4)	73.5 (10.1)	.828
[c]CASI Exam 5, M (SD)	92.3 (5.9)	92.1 (5.3)	.865
[d]CASI Exam 6, M (SD)	93.6 (4.5)	90.3 (8.4)	.002*
[e]CASI change (5 to 6), M (SD)	1.4 (5.0)	−1.8 (6.8)	.001*
PiB SUVR, M (SD)	1.11 (0.05)	1.71 (0.27)	<.001*
[f]WMH vol. (% ICV), median (IQR)	0.19 (0.10, 0.42)	0.33 (0.14, 0.72)	.025*
[d]Cort. thick., mm, M (SD)	2.69 (0.12)	2.65 (0.13)	.037*

Abbreviations: Aβ, β-amyloid; ASCVD, atherosclerotic cardiovascular disease risk estimate from the pooled cohort equation; FSRP, Framingham stroke risk profile; CAIDE, cardiovascular risk factors, aging and incidence of dementia risk score; SBP, systolic blood pressure; DBP, diastolic blood pressure; CASI, Cognitive Abilities Screening Instrument; PiB SUVR, Pittsburgh compound B Standardized Uptake Value Ratio; Cort. thick., cortical thickness; WMH vol., white matter hyperintensity volume; ICV, intracranial volume. *Statistically significant at $P < .05$. Note: 5 participants (3 Aβ-, 2 Aβ+) did not have cognitive status available

[a]Aβ- n = 99; Aβ+ n = 55.
[b]Aβ- n = 101; Aβ+ n = 57.
[c]Aβ- n = 99; Aβ+ n = 57.
[d]Aβ- n = 102; Aβ+ n = 56.
[e]Aβ- n = 99; Aβ+ n = 56.
[f]Aβ- n = 101; Aβ+ n = 56.

We observed differences between AA and White participants in antecedent VRF scores, but these preliminary single-site results also suggest that AA group members were no more likely to be Aβ+ or show differences in WMH and cortical thickness in AD-prone temporal regions than White participants. Further, we found not only that all 3 VRF scores were strongly associated with MRI biomarkers of WMH and temporal lobe cortical thickness, but also that CAIDE and FSRP were associated with Aβ PET. VRFs showed a more complex pattern of associations with cognitive performance, described in more detail below. Finally, we found that VRF scores, WMH, cortical thickness, and Aβ deposition were significant predictors of cognitive performance and cognitive decline, even when controlling for covariates.

4.1 | Aβ group differences

Aβ positivity in a diverse older adult sample was associated with expected predictors of age and *APOE*-ε4,[38,39] with reduced cognitive performance at time of imaging and greater retrospective cognitive decline,[40] and with greater WMH volume and lower temporal lobe cortical thickness.[41] This set of regions is usually susceptible to age-related neurodegenerative disease, including typical amnestic AD and limbic-predominant age-related TDP-43-encephalopathy (LATE). These results in a diverse sample confirm and extend previous research (mostly conducted in White populations of European descent) that found similar results.[39–41] Aβ positivity was associated with greater

TABLE 2 Participant measures by race

	African-American	White	*P*
n (%)	79 (49.7)	80 (50.3)	
Age, Exam 1, M (SD)	55.8 (6.8)	55.7 (6.5)	.945
Age, Exam 6, M (SD)	71.5 (6.7)	71.8 (6.6)	.810
Sex (M/F)	50/29	38/42	.045*
Years of education, M (SD)	14.9 (2.7)	15.1 (2.8)	.729
[a]Cognitive status			.618
Normal, n (%)	49 (62.8)	57 (72.2)	
MCI, n (%)	24 (30.8)	18 (22.8)	
Dementia, n (%)	3 (3.9)	3 (3.8)	
APOE-ε4, (+/-/missing)	31/45/3	22/55/3	.283
% ASCVD, M (SD)	8.76 (6.8)	5.45 (4.5)	<.001*
[b]% FSRP, M (SD)	2.33 (2.4)	1.78 (1.8)	.096
CAIDE, M (SD)	7.25 (2.3)	6.55 (2.2)	.051
SBP, M (SD)	133.0 (21.9)	121.0 (17.4)	<.001*
DBP, M (SD)	75.7 (9.2)	71.9 (11.0)	.020*
[c]CASI Exam 5, M (SD)	89.7 (6.2)	94.6 (3.7)	<.001*
[d]CASI Exam 6, M (SD)	90.6 (7.2)	94.3 (4.8)	<.001*
[e]CASI change (5 to 6), M (SD)	0.8 (6.8)	−0.4 (4.9)	.224
PiB SUVR, M (SD)	1.27 (0.28)	1.39 (0.37)	.029*
Aβ+, *n* (%)	25 (31.7)	32 (40.0)	.272
[f]WMH vol. (% ICV), median (IQR)	0.21 (0.11, 0.55)	0.23 (0.10, 0.55)	.979
[d]Cort. Thick., mm, M (SD)	2.66 (0.13)	2.69 (0.12)	.143

Abbreviations: Aβ, β-amyloid; ASCVD, atherosclerotic cardiovascular disease risk estimate from the pooled cohort equation; FSRP, Framingham stroke risk profile; CAIDE, cardiovascular risk factors, aging, and incidence of dementia risk score; SBP, systolic blood pressure; DBP, diastolic blood pressure; CASI, Cognitive Abilities Screening Instrument; PiB SUVR, Pittsburgh compound B Standardized Uptake Value Ratio; Cort. thick., Cortical thickness; WMH vol., white matter hyperintensity volume; ICV, intracranial volume. *Statistically significant at $P < .05$

[a]White n = 78; African-American n = 76.
[b]White n = 80; African-American n = 78.
[c]White n = 80; African-American n = 76.
[d]White n = 79; African-American n = 79.
[e]White n = 79; African-American n = 76.
[f]White n = 79; African-American n = 78.

prevalence of cognitive impairment and dementia, and higher FSRP and CAIDE as well.

4.2 | Group differences by race

We found that AA participants had elevated ASVCD (expected, as race is a component of the ASCVD score), as well as a marginal difference in CAIDE. In contrast with the Atherosclerosis Risk in Communities (ARIC) study, which examined brain Aβ in AA and White cohorts from two different geographic regions, we did not find group differences by race in Aβ positivity in our single-site sample, suggesting that AA are not necessarily more likely to be Aβ+;[38] indeed, we observed that AA had lower overall PiB SUVR, which warrants further investigation. These findings of group differences by race in vascular burden inimical toward the brain in a single-site cohort in the southeastern US, in the absence of neuroimaging differences, suggest complex late-life relationships among vascular, imaging, and cognitive measures in a diverse older adult sample. We again note that in this analysis, we interpret race as a social, and not biological, construct. We do not know (it was not tested) whether these group differences by race were due to social determinants of health or to intrinsic differences.[42] A more extensive analysis of MESA VRF, neuroimaging, and cognitive testing data with respect to social determinants of health is planned for future work.

4.3 | Associations among VRF scores, imaging biomarkers, and cognition

Higher baseline VRF scores were consistently associated with increased WMH volumes and reduced cortical thickness across

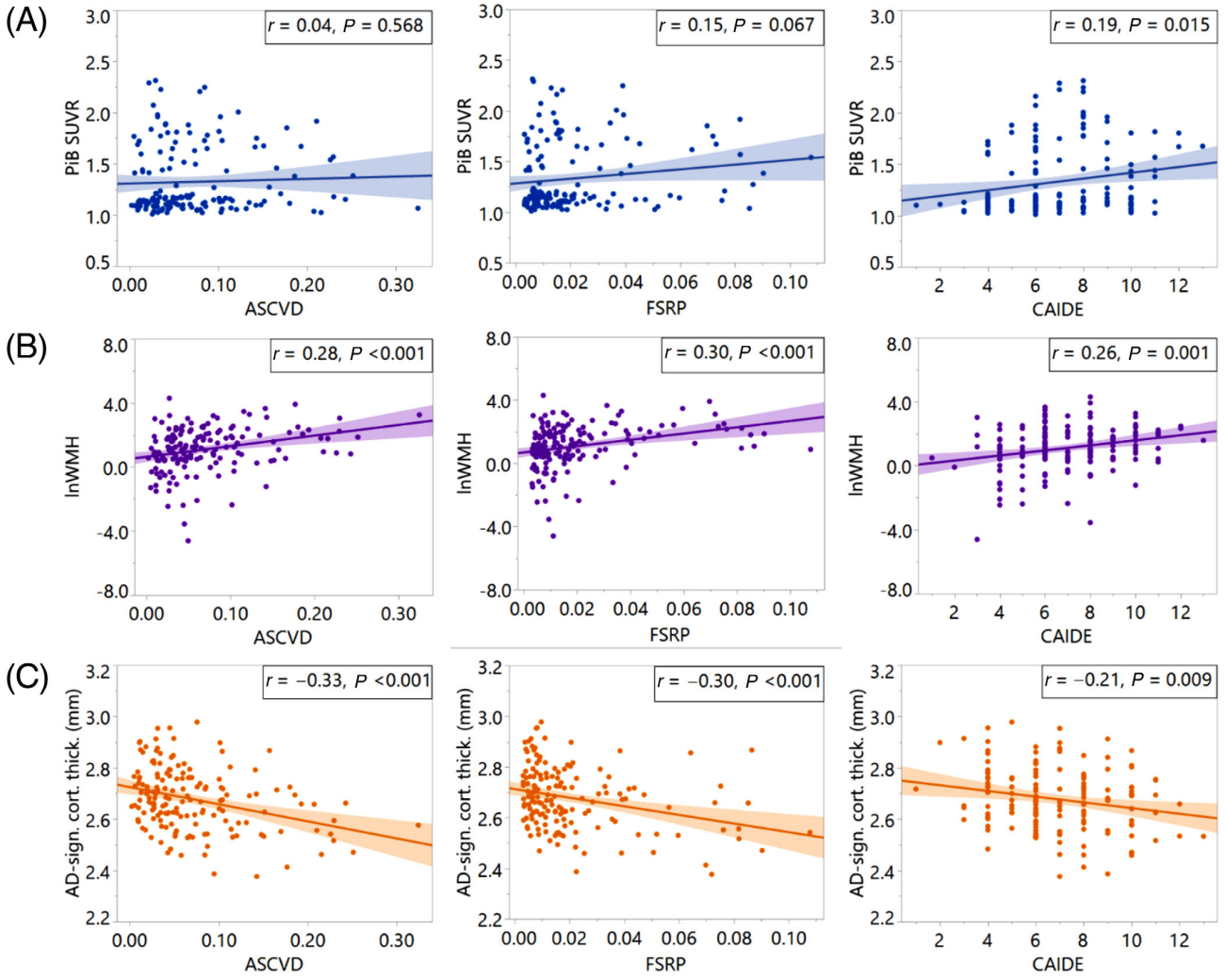

FIGURE 1 Vascular risk factor (VRF)-imaging scatterplots. VRFs (x-axes) versus (A) Global PiB SUVR, (B) log-norm. WMH volume, and (C) temporal lobe cortical thickness (y-axes). ASCVD, atherosclerotic cardiovascular disease risk estimate from the pooled cohort equation; FSRP, Framingham stroke risk profile; CAIDE, cardiovascular risk factors, aging and incidence of dementia risk score; PiB SUVR, Pittsburgh compound B Standardized Uptake Value Ratio; lnWMH, log-normalized white matter hyperintensity volume; AD-sign. cort. thick., temporal lobe cortical thickness

groups; interestingly, elevated FSRP and CAIDE were associated with greater Aβ as well. The association between CAIDE and brain Aβ deposition may be related to the inclusion of education and *APOE*, both known dementia risk factors, in the CAIDE score. The association between Aβ and FSRP, which includes prevalent CVD and atrial fibrillation, suggests these factors may be important avenues to explore with respect to their relationship to Aβ. As expected, VRF scores were associated with WMH volume, a well-known biomarker of SVD.[9,10] Further, baseline VRF scores were associated with lower cortical thickness, indicating elevated mid- to late-life VRFs can predict neurodegeneration approximately 16 years later. There were significant interactions by race with respect to cortical thickness as well, such that associations with ASCVD and FSRP were stronger in White than in AA participants.

We also looked at predictors of cognitive performance and change over a 6-year period. We found Aβ predicted lower Exam 6 CASI performance and greater decline, and measures of WMH and cortical thickness were significantly related to CASI change. However, we note that cross-sectional associations between PiB SUVR and cognition appeared to be driven by those who were classified with MCI or dementia at Exam 6. Further, we found no significant associations of VRF scores with Exam 5 CASI scores, but increases in all three VRF scores were associated with worse Exam 6 CASI performance , with the effect of CAIDE driven primarily by the AA group. Elevated FSRP and CAIDE also related to greater CASI decline. These results suggest complex links between stroke risk and cognitive decline in this cohort free from stroke, and imply that controlling VRFs may be a potent mechanism for preventing cognitive decline in preclinical stages of AD.

TABLE 3 VRF-Imaging associations

	t	P	Race interaction P
PiB SUVR (n = 159)			
ASCVD[a]	0.37	.716	.606
FSRP[b,d]	2.48	.014*	.254
CAIDE[c]	2.89	.004*	.666
WMH Volume (n = 157)			
ASCVD[a]	3.71	<.001*	.157
FSRP[b,d]	3.94	<.001*	.183
CAIDE[c]	3.44	<.001*	.945
Cort. Thick. (n = 158)			
ASCVD[a]	−4.28	<.001*	.004*
FSRP[b,d]	−3.69	<.001*	.013*
CAIDE[c]	−2.47	.014*	.975

Note: all VRFs include age, sex, and systolic blood pressure. VRF components listed in Supplementary Table 1.
Abbreviations: VRF, vascular risk factor; ASCVD, atherosclerotic cardiovascular disease risk estimate from the pooled cohort equation; FSRP, Framingham stroke risk profile; CAIDE, cardiovascular risk factors, aging, and incidence of dementia risk score; PiB SUVR, Pittsburgh compound B Standardized Uptake Value Ratio; WMH, white matter hyperintensity; Cort. thick., cortical thickness.
[a]Adjusted for education and *APOE-ε4* carriage.
[b]Adjusted for education, race, and *APOE-ε4* carriage.
[c]Adjusted for race.
[d]One outlier excluded.
*Significant at $P < .05$.

TABLE 4 VRF-CASI associations

	t	P	Race interaction P
CASI Exam 5 (n = 156)			
ASCVD[a]	−1.31	.191	.987
FSRP[b,d]	0.51	.611	.610
CAIDE[c]	−1.73	.085	.176
CASI Exam 6 (n = 158)			
ASCVD[a]	−3.01	.003*	.943
FSRP[b,d]	−2.39	.018*	.901
CAIDE[c]	−4.16	<.001*	.037*
CASI Change (n = 155)			
ASCVD[a]	−1.70	.091	.984
FSRP[b,d]	−2.74	.007*	.789
CAIDE[c]	−2.63	.010*	.401

Note: all VRFs include age, sex, and systolic blood pressure. VRF components listed in Supplementary Table 1.
Abbreviations: VRF, vascular risk factor; ASCVD, atherosclerotic cardiovascular disease risk estimate from the pooled cohort equation; FSRP, Framingham stroke risk profile; CAIDE, cardiovascular risk factors, aging, and incidence of dementia risk score; CASI, Cognitive Abilities Screening Instrument.
[a]Adjusted for education and *APOE-ε4* carriage.
[b]Adjusted for education, race, and *APOE-ε4* carriage.
[c]Adjusted for race.
[d]One outlier excluded.
*Significant at $P < .05$.

TABLE 5 Imaging-CASI associations

	n	t[a]	P	Race interaction P
CASI Exam 5				
PiB SUVR	156	−0.13	.897	.132
WMH Volume	154	1.15	.252	.695
Cort. Thick.	155	−0.48	.631	.593
CASI Exam 6				
PiB SUVR	158	−2.47	.015*	.779
WMH Volume	156	−1.45	.149	.308
Cort. Thick.	157	1.66	.099	.859
CASI Change				
PiB SUVR	155	−2.23	.028*	.126
WMH Volume	153	−2.35	.020*	.221
Cort. Thick.	154	1.99	.048*	.859

Abbreviations: PiB SUVR, Pittsburgh compound B Standardized Uptake Value Ratio; WMH, white matter hyperintensity; Cort. thick., cortical thickness; CASI, Cognitive Abilities Screening Instrument.
[a]Adjusted for age, race, sex, education, and *APOE-ε4* carriage.
*Significant at $P < .05$.

4.4 | Implications, limitations, and future directions

Our results were primarily in agreement with previous studies. We found FSRP, a known predictor of 10-year risk for vascular brain injury,[43] to be associated with lower cortical thickness, higher WMH, and cognitive decrements, consistent with previous work.[27–29,44] As initially demonstrated in the FINGER study, we found CAIDE to be associated with higher WMH and lower cortical thickness; however, in contrast, we also observed CAIDE to be significantly associated with elevated brain Aβ, in a diverse larger sample.[18]

There are some limitations in the present research. We chose to examine a series of global and composite measures in order to canvass the scope of late-life associations among VRFs, imaging biomarkers, and cognition; future efforts will examine more specific clinical and subclinical VRFs, and regional and voxelwise patterns of imaging measures. Repeated detailed cognitive testing and neuroimaging are ongoing in this study, and will enable more specific cognitive domains to be evaluated in future research. Finally, given the sample size, a proper examination of interactions by sex in multivariable models, as well as an analysis of individual VRFs examined independently, are beyond the scope of this analysis.

In this multimodal, longitudinal study, we found differential associations among mid- to late-life VRF scores, brain imaging biomarkers of Aβ pathology, SVD, and neurodegeneration, and cognitive performance and longitudinal cognitive decline. Limited previous research has found differential VRF-imaging associations for different imaging modalities,[18] and growing evidence suggests that controlling VRFs may be beneficial to cognition.[17] The examination of unique associations of VRFs with imaging biomarkers in a diverse study may illuminate targets for protecting brain health and cognition among diverse older adults.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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Gov. Ex. 175

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

SAN JOSE DIVISION

UNITED STATES OF AMERICA,	)	CR-20-00371 WHA
	)	
PLAINTIFF,	)	SAN JOSE, CALIFORNIA
	)	
VS.	)	OCTOBER 15, 2020
	)	
ROBERT T. BROCKMAN,	)	PAGES 1-33
	)	
DEFENDANT.	)	
	)	

TRANSCRIPT OF ZOOM PROCEEDINGS
BEFORE THE HONORABLE NATHANAEL COUSINS
UNITED STATES MAGISTRATE JUDGE

A P P E A R A N C E S:

FOR THE PLAINTIFF: UNITED STATES ATTORNEY'S OFFICE
BY: MICHAEL G. PITMAN
150 ALMADEN BOULEVARD, SUITE 900
SAN JOSE, CALIFORNIA 95113

FOR THE DEFENDANT: JONES DAY
BY: NEAL J. STEPHENS
1755 EMBARCADERO ROAD
PALO ALTO, CALIFORNIA 94303

BY: JASON S. VARNADO
717 TEXAS, SUITE 3300
HOUSTON, TEXAS 77002

PRETRIAL OFFICER: JAY CHRISTIAN

OFFICIAL COURT REPORTER: LEE-ANNE SHORTRIDGE, CSR, CRR
CERTIFICATE NUMBER 9595

PROCEEDINGS RECORDED BY MECHANICAL STENOGRAPHY
TRANSCRIPT PRODUCED WITH COMPUTER

SAN JOSE, CALIFORNIA OCTOBER 15, 2020

P R O C E E D I N G S

(ZOOM PROCEEDINGS CONVENED AT 12:17 P.M.)

THE COURT: ALL RIGHT. REJOINING, GOOD AFTERNOON.

THE CLERK: CALLING 3:20-0371, UNITED STATES VERSUS ROBERT T. BROCKMAN.

COUNSEL, PLEASE STATE YOUR APPEARANCES FOR THE RECORD.

MR. PITMAN: GOOD AFTERNOON, YOUR HONOR.

MICHAEL PITMAN FOR THE UNITED STATES.

THE COURT: GOOD AFTERNOON.

MR. STEPHENS: GOOD AFTERNOON, YOUR HONOR.

IT'S NEAL STEPHENS FROM JONES DAY IN PALO ALTO APPEARING SPECIALLY FOR MR. BROCKMAN FOR TODAY'S PURPOSES. WITH ME AS WELL IS MY PARTNER FROM HOUSTON, JASON VARNADO.

AND AS YOU CAN SEE, MR. BROCKMAN IS ALSO PRESENT AND BEFORE THE COURT FROM HOUSTON AND CONSENTS TO THE VIDEO NATURE OF THE APPEARANCE.

THE COURT: THANK YOU.

AND IF I CAN JUST HAVE MR. BROCKMAN IDENTIFY HIMSELF AND CONFIRM THAT HE'S ABLE TO SEE AND HEAR US SO FAR.

I'M JUDGE COUSINS. I'M THE DUTY JUDGE TODAY.

THE DEFENDANT: YES, SIR.

MY NAME IS ROBERT T. BROCKMAN, AND I CAN SEE AND HEAR WITH THE MAGICAL SOFTWARE SET UP HERE.

THE COURT: VERY GOOD. THANK YOU FOR BEING WITH US

TODAY.

WE ARE MAKING A RECORDING AND A TRANSCRIPTION OF TODAY'S HEARING.

ALSO WITH US FROM U.S. PRETRIAL SERVICES IS OFFICER CHRISTIAN, WHO'S ALSO ON THE SCREEN.

THE PURPOSE OF COURT TODAY, MR. BROCKMAN, IS TO ADVISE YOU OF YOUR RIGHTS, TO ALERT YOU TO THE CHARGES FILED AGAINST YOU IN AN INDICTMENT THAT WAS RETURNED BY THE GRAND JURY IN THIS DISTRICT, AND ALSO TO DISCUSS CONDITIONS OF RELEASE OR DETENTION AND TO SET SOME FURTHER DATES IN THE CASE.

THIS CASE HAS BEEN ASSIGNED TO DISTRICT COURT JUDGE WILLIAM ALSUP IN SAN FRANCISCO.

THE INDICTMENT WAS ORIGINALLY FILED UNDER SEAL.

MR. PITMAN, DOES THE GOVERNMENT MOVE TO UNSEAL THE INDICTMENT?

MR. PITMAN: YES, YOUR HONOR.

THE COURT: ALL RIGHT. THAT REQUEST IS GRANTED. THE INDICTMENT IS UNSEALED.

AND MR. STEPHENS AND MR. VARNADO, HAVE YOU RECEIVED A COPY OF IT?

MR. STEPHENS: WE GOT IT JUST EARLIER THIS WEEK, YOUR HONOR.

THE COURT: ALL RIGHT. THANK YOU.

MR. BROCKMAN, NOW LET ME ADVISE YOU OF YOUR RIGHTS.

YOU HAVE THE RIGHT TO REMAIN SILENT. ANY WORDS THAT YOU

SAY CAN BE USED AGAINST YOU. IF YOU'VE ALREADY SPOKEN WITH A GOVERNMENT AGENT, YOU DO NOT NEED TO SAY ANYTHING MORE. IF YOU START TO MAKE A STATEMENT, YOU MAY STOP IMMEDIATELY.

YOU HAVE THE RIGHT TO AN ATTORNEY AT EVERY STAGE OF THE CASE, AND IF YOU CANNOT AFFORD AN ATTORNEY, THE COURT WOULD APPOINT ONE FOR YOU AT NO COST TO YOU.

I UNDERSTAND THAT YOU HAVE RETAINED COUNSEL AND THEY'RE APPEARING WITH YOU TODAY, BUT IF CERTAIN THINGS CHANGE AND YOU WISH THE COURT TO APPOINT AN ATTORNEY FOR YOU, YOU'LL LET US KNOW AND WE'LL EVALUATE WHETHER YOU QUALIFY FOR COUNSEL.

DO YOU UNDERSTAND THAT RIGHT?

THE DEFENDANT: YES, I DO. THANK YOU, SIR.

THE COURT: ALL RIGHT. THANK YOU.

NEXT I'D LIKE TO ASK MR. PITMAN TO SUMMARIZE THE CHARGES IN THE INDICTMENT AGAINST MR. BROCKMAN AND TO ADVISE HIM OF THE MAXIMUM PENALTIES IF HE WERE CONVICTED. HE IS PRESUMED INNOCENT OF THE CHARGES. THIS IS JUST TO ADVISE HIM OF THE CHARGES HE FACES.

MR. PITMAN, YOU MAY PROCEED.

MR. PITMAN: THANK YOU, YOUR HONOR.

THE DEFENDANT HAS BEEN CHARGED WITH ONE COUNT OF CONSPIRACY IN VIOLATION OF 18 UNITED STATES CODE, SECTION 371.

THE MAXIMUM PENALTIES ARE FIVE YEARS IN PRISON; A $250,000 FINE; THREE YEARS SUPERVISED RELEASE; AND A $100 SPECIAL ASSESSMENT.

THE DEFENDANT HAS ALSO BEEN CHARGED WITH SEVEN COUNTS OF TAX EVASION IN VIOLATION OF 26 UNITED STATES CODE, SECTION 7201.

THE MAXIMUM PENALTIES FOR EACH COUNT ARE FIVE YEARS IN PRISON; A $250,000 FINE; THREE YEARS SUPERVISED RELEASE; A $100 SPECIAL ASSESSMENT; AND THE COSTS OF PROSECUTION ARE AUTHORIZED AS A PENALTY FOR THOSE COUNTS.

THE DEFENDANT HAS ALSO BEEN CHARGED WITH SIX COUNTS OF FBAR VIOLATIONS, FOREIGN BANK ACCOUNT REPORTING VIOLATIONS, IN VIOLATION OF 31 UNITED STATES CODE, SECTIONS 5314 AND 5322(B).

THE MAXIMUM PENALTIES FOR EACH COUNT ARE TEN YEARS IN PRISON; A $500,000 FINE; THREE YEARS SUPERVISED RELEASE; AND A $100 SPECIAL ASSESSMENT.

THE DEFENDANT HAS ALSO BEEN CHARGED WITH 20 COUNTS OF WIRE FRAUD AFFECTING A FINANCIAL INSTITUTION IN VIOLATION OF 18 UNITED STATES CODE, SECTION 1343.

THE MAXIMUM PENALTY FOR EACH COUNT IS 30 YEARS IN PRISON; A $1 MILLION FINE; FIVE YEARS SUPERVISED RELEASE; AND A $100 SPECIAL ASSESSMENT.

THE DEFENDANT HAS ALSO BEEN CHARGED WITH TWO COUNTS OF CONCEALMENT MONEY LAUNDERING IN VIOLATION OF 18 UNITED STATES CODE, SECTION 1956(A)(1)(B)(1).

THE MAXIMUM PENALTIES FOR EACH COUNT ARE 20 YEARS IN PRISON; A $500,000 FINE OR TWICE THE GROSS GAIN OR LOSS, WHICHEVER IS GREATER; THREE YEARS SUPERVISED RELEASE; AND A

$100 SPECIAL ASSESSMENT.

THE DEFENDANT HAS ALSO BEEN CHARGED WITH TWO COUNTS OF TAX EVASION MONEY LAUNDERING IN VIOLATION OF 18 UNITED STATES CODE, SECTION 1956(A)(1)(A)(2).

THE MAXIMUM PENALTIES FOR EACH COUNT ARE 20 YEARS IN PRISON; A $500,000 FINE OR TWICE THE GROSS GAIN OR LOSS, WHICHEVER IS GREATER; THREE YEARS SUPERVISED RELEASE; AND A $100 SPECIAL ASSESSMENT.

THE DEFENDANT HAS ALSO BEEN CHARGED WITH ONE COUNT OF INTERNATIONAL CONCEALMENT MONEY LAUNDERING IN VIOLATION OF 18 UNITED STATES CODE, SECTION 1956(A)(2)(B)(1).

THE MAXIMUM PENALTY FOR THIS COUNT IS 20 YEARS IN PRISON; A $500,000 FINE OR TWICE THE GROSS GAIN OR LOSS, WHICHEVER IS GREATER; THREE YEARS SUPERVISED RELEASE; AND A $100 SPECIAL ASSESSMENT.

THE DEFENDANT HAS ALSO BEEN CHARGED WITH ONE COUNT OF EVIDENCE TAMPERING IN VIOLATION OF 18 UNITED STATES CODE, SECTION 1512(B)(2)(B).

THE MAXIMUM PENALTY FOR THIS COUNT IS 20 YEARS IN PRISON; A $250,000 FINE; THREE YEARS SUPERVISED RELEASE; AND A $100 SPECIAL ASSESSMENT.

THE DEFENDANT HAS ALSO BEEN CHARGED WITH ONE COUNT OF DESTRUCTION OF EVIDENCE IN VIOLATION OF 18 UNITED STATES CODE, SECTION 1512(C)(1).

THE MAXIMUM PENALTY FOR THIS COUNT IS 20 YEARS IN PRISON;

A $250,000 FINE; THREE YEARS SUPERVISED RELEASE; AND A $100 SPECIAL ASSESSMENT.

THE COURT: THANK YOU.

MR. STEPHENS, WOULD YOU WAIVE A MORE DETAILED READING OF THOSE CHARGES?

MR. STEPHENS: YES, YOUR HONOR.

FOR TODAY'S PURPOSES, JUST FOR THE COURT'S BENEFIT, WE -- IT'S A LENGTHY, COMPLEX INDICTMENT. WE JUST RECEIVED IT. WE HAVE COVERED IT, AS YOU CAN APPRECIATE IT, WITH OUR CLIENT. WE ARE PREPARED TO WAIVE READING FOR SURE AND ENTER A PLEA OF NOT GUILTY TO ALL COUNTS AND A DENIAL OF ALL THE FORFEITURE ALLEGATIONS.

THE COURT: THANK YOU. THE NOT GUILTY PLEA IS ENTERED.

MR. PITMAN, A REMINDER TO YOU, THE INDICTMENT DOES ALLEGE CRIME VICTIMS, IT IDENTIFIES VICTIMS OF THE WIRE FRAUD AFFECTING A FINANCIAL INSTITUTION AT THE LEAST, SO A REMINDER THAT THE GOVERNMENT HAS AN OBLIGATION TO NOTIFY ANY CRIME VICTIMS TO ALLOW THEM TO HAVE NOTICE OF THE PROCEEDINGS, TO CONSULT WITH GOVERNMENT COUNSEL, AND TO PARTICIPATE IN CERTAIN PROCEEDINGS BEFORE THIS COURT. SO JUST A REMINDER THAT YOU HAVE THAT OBLIGATION TO GIVE THEM THAT NOTICE AND OPPORTUNITY TO PARTICIPATE IN THE PROCEEDINGS.

NEXT TOPIC IS CONDITIONS OF RELEASE OR DETENTION.

MR. PITMAN, WHAT IS THE GOVERNMENT'S MOTION AS TO

MR. BROCKMAN'S CONDITIONS DURING THE CASE?

MR. PITMAN: THE GOVERNMENT IS NOT MOVING FOR DETENTION.

IT WOULD BE OUR PROPOSAL THAT THE DEFENDANT BE RELEASED ON CONDITIONS, INCLUDING A $1 MILLION SECURED BOND, AND THAT HIS TRAVEL BE RESTRICTED TO NINE DISTRICTS, IN ADDITION TO THE STANDARD TERMS, AND I DO NOT BELIEVE THAT THOSE TERMS ARE AGREED.

THE COURT: ALL RIGHT. SO THAT -- WHAT YOU JUST GAVE ME IS WHAT THE GOVERNMENT'S MOTION IS, AND YOU EXPECT THAT THE DEFENSE HAS A DIFFERENT VIEW.

AT THIS MOMENT, HAS HE HAD ANY COMMUNICATIONS WITH PRETRIAL SERVICES ABOUT WHAT MIGHT BE APPROPRIATE CONDITIONS?

MR. STEPHENS: HE HAS NOT, YOUR HONOR.

THE COURT: AND HAS HE BEEN BOOKED BY ANY U.S. MARSHALS ON THE CHARGE?

MR. STEPHENS: NO, SIR, YOUR HONOR, BUT IT WAS OUR INTENTION TO DO THAT.

WE -- FOR THE COURT'S BENEFIT, WE SPOKE TWICE WITH GOVERNMENT COUNSEL AND WE HAVE REACHED AGREEMENT IN WORKING THROUGH THE COURT'S ORDER SETTING CONDITIONS OF RELEASE, EXCEPT FOR ONE LAST ITEM, AND THAT IS THE TERMS OF BAIL. WE THINK IT'S INAPPROPRIATE TO HAVE A CASH BAIL OR CORPORATE SURETY BOND HERE.

I CAN WALK YOU THROUGH THE OTHER TERMS THAT WE SPOKE WITH

MR. PITMAN ABOUT EARLIER THIS WEEK. THOSE ARE THE FIRST THREE ON YOUR SHEET HERE, YOUR HONOR, THAT THE DEFENDANT WILL OBVIOUSLY APPEAR, MR. BROCKMAN WILL APPEAR; HE WILL NOT COMMIT ANY FEDERAL OR STATE CRIME; HE SHALL NOT HARASS, THREATEN, INTIMIDATE, ET CETERA.

WE ALSO AGREE THAT HE WILL REPORT IN PERSON TO PRETRIAL SERVICES. HE CAN DO SO THERE IN HOUSTON. WE HAVE ALREADY REACHED OUT TO CONTACT THAT OFFICE AND ARE WILLING TO DO THAT IN AN EXPEDITIOUS FASHION.

AND HE, FOR THE COURT'S BENEFIT, HAS ALREADY SURRENDERED TO US, AT THE GOVERNMENT'S REQUEST, HIS PASSPORT.

SO THOSE ARE --

THE COURT: SORRY TO INTERRUPT. SO THE -- IF YOU'RE NOT DONE, I'LL STOP SO YOU CAN FINISH.

MR. STEPHENS: OH, NO. GO AHEAD.

THE COURT: SO THE PART THAT'S DISAGREED UPON IS THE AMOUNT AND SECURITIZATION OF THE BOND? IS THAT FAIR?

MR. STEPHENS: YEAH, THAT'S CORRECT, YOUR HONOR.

WE HAD DISAGREEMENT OVER THE JURISDICTIONS THAT HE COULD TRAVEL IN WITHIN THE UNITED STATES, BUT THE BAIL ISSUE IS BY FAR THE MOST IMPORTANT ONE TO US. WE WILL AGREE WITH THE GOVERNMENT ON THE JURISDICTIONS THAT THEY HAVE PROPOSED, AND FOR A.U.S.A. PITMAN'S BENEFIT, I'LL READ THEM IN, AND IF I DON'T HAVE THEM RIGHT, PLEASE CORRECT ME.

THE COURT: BEFORE YOU READ ANYTHING IN, IS THERE A

WRITING? IF YOU'VE GOT A DRAFT BOND FORM, HAVE YOU GOT THAT WRITTEN DOWN AND YOU CAN SEND IT TO ME?

MR. STEPHENS: I WISH I DID, YOUR HONOR. I APOLOGIZE, BUT I DO NOT.

THE COURT: ALL RIGHT. THEN YOU CAN TELL ME WHAT THEY ARE.

MR. STEPHENS: SURE.

THE DISTRICTS ARE THE SOUTHERN DISTRICT OF TEXAS; THE DISTRICT OF COLORADO; THE NORTHERN DISTRICT OF CALIFORNIA; THE SOUTHERN DISTRICT OF NEW YORK; THE D.C. AREA DISTRICTS, YOUR HONOR, THE DISTRICT OF COLUMBIA AND THE EASTERN DISTRICT OF VIRGINIA; AND THEN TWO DISTRICTS IN OHIO, THE SOUTHERN DISTRICT OF OHIO AND THE NORTHERN DISTRICT OF OHIO.

AND THOSE REPRESENT, YOUR HONOR, AREAS WHERE HE EITHER RESIDES OR HIS LAWYERS ARE OR HIS COMPANY IS.

THE COURT: ALL RIGHT. LET ME JUST SEGUE ON THAT ONE.

MR. PITMAN, WHAT'S THE BASIS FOR HAVING A TRAVEL RESTRICTION TO NINE DIFFERENT DISTRICTS AND WHAT'S THE CONCERN?

MR. PITMAN: WELL, IT'S THE -- THE CONCERN IS THAT THE DEFENDANT POSES A RISK OF FLIGHT, YOUR HONOR.

THE COURT: ALL RIGHT. AND ARTICULATE FOR ME WHAT ESTABLISHES THAT CONCERN.

MR. PITMAN: OKAY. SO OBVIOUSLY THERE ARE STANDARD -- AND THIS IS -- I'LL ALSO INCORPORATE THE ARGUMENT I

WOULD MAKE WITH RESPECT TO THE SECURITY ON THE BOND, YOUR HONOR.

THE BAIL REFORM ACT ANTICIPATES THAT WE'LL IMPOSE -- THE COURT WILL IMPOSE THE LEAST RESTRICTIVE CONDITIONS NECESSARY TO REASONABLY ASSURE THAT THE DEFENDANT WILL APPEAR AND GIVES US A FRAMEWORK FOR ANALYZING THE POTENTIAL EFFICACY OF PROPOSED CONDITIONS, INCLUDING THE NATURE AND CIRCUMSTANCES OF THE OFFENSE, THE WEIGHT OF THE EVIDENCE AGAINST THE DEFENDANT, THE HISTORY AND CHARACTERISTICS OF THE DEFENDANT, AND THE DANGER TO THE COMMUNITY.

IN THIS CASE, THESE FACTORS DO SUPPORT IMPOSING CONDITIONS ON THE DEFENDANT'S RELEASE, INCLUDING A SIGNIFICANT CASH BOND, AND RESTRICTING HIS TRAVEL ONLY TO DISTRICTS THAT HE NEEDS TO TRAVEL TO IN ORDER TO LITIGATE THIS CASE. HE'S GOT ATTORNEYS ACROSS THE COUNTRY AND THE GOVERNMENT HAS AGREED THAT THERE'S NO PROBLEM HAVING HIM TRAVEL TO THE DISTRICTS IN WHICH HE NEEDS TO FOR HIS DEFENSE.

THERE'S ALSO TWO DISTRICTS IN OHIO THAT HE NEEDS TO TRAVEL TO IN ORDER TO DO HIS WORK. HE'S THE CEO OF A COMPANY WITH THOUSANDS OF EMPLOYEES, SO WE'VE AGREED TO THAT.

SO I'LL GO THROUGH THE FACTORS QUICKLY, YOUR HONOR.

THE NATURE AND CIRCUMSTANCES OF THE OFFENSE ARE SERIOUS. WE'VE JUST GONE THROUGH THE PENALTIES.

THE INDICTMENT, IN CASE THE COURT HASN'T READ THE WHOLE THING, ALLEGES AN EVASION, A TAX EVASION OF OVER $2 BILLION.

IT ALSO ALLEGES THAT THE DEFENDANT DEFRAUDED HIS BUSINESS PARTNERS AND LAUNDERED THE PROCEEDS, WHICH WERE IN EXCESS OF $60 MILLION, AND IT ALSO ALLEGES THAT THE DEFENDANT WAS INVOLVED IN PHYSICALLY DESTROYING EVIDENCE THAT IS PERTINENT TO THE CASE.

SO THAT -- IF THOSE CHARGES ARE ALL PROVEN, THIS COULD BE THE MOST SIGNIFICANT INCOME TAX EVASION CASE AGAINST AN INDIVIDUAL IN UNITED STATES HISTORY.

WITH RESPECT TO THE WEIGHT OF THE EVIDENCE AGAINST THE DEFENDANT, I KNOW THAT'S THE LEAST IMPORTANT FACTOR, AS I'M SURE THE COURT WILL REMIND ME.

I'LL JUST SAY THAT THE INDICTMENT IN THIS CASE IS 40 PAGES LONG. IT INCORPORATES 39 COUNTS COVERING CRIMINAL CONDUCT SPANNING TWO DECADES. THE INVESTIGATION, AS ALLEGED IN THE INDICTMENT, HAS BEEN ONGOING FOR AT LEAST FOUR YEARS. SO I WOULD JUST SAY THE INDICTMENT SPEAKS FOR ITSELF WITH RESPECT TO THE STRENGTH OF THE EVIDENCE AGAINST THE DEFENDANT.

THE HISTORY AND CHARACTERISTICS OF THE DEFENDANT ARE REALLY WHAT I'D LIKE TO DRAW THE COURT'S ATTENTION TO.

I THINK THE RECORD DEMONSTRATES BEYOND A PREPONDERANCE STANDARD THAT THE DEFENDANT DOES POSE A FLIGHT RISK, AND THERE ARE THREE THINGS I'D POSIT AS GOOD EVIDENCE OF THAT RISK, YOUR HONOR.

THE FIRST IS THIS IS A VERY SERIOUS CASE. AS I SAID BEFORE, IF THE DEFENDANT WERE TO SUFFER CONVICTIONS ON EVEN A

FEW OF THE COUNTS ALLEGED IN THIS INDICTMENT, HE COULD FACE A SEVERE INCARCERATIVE PENALTY, WHICH IS OBVIOUSLY A CONCERN FOR ANY DEFENDANT, BUT ESPECIALLY ONE WHO'S 79 YEARS OLD.

THE SECOND CONCERN WE HAVE IS THAT THE DEFENDANT HAS THE MEANS TO FLEE, AND THOSE ARE EMBODIED IN TWO DIFFERENT CATEGORIES OF EVIDENCE, I SUPPOSE.

THE FIRST IS THAT THE DEFENDANT, FROM OUR PERSPECTIVE, HAS VIRTUALLY UNLIMITED FINANCIAL RESOURCES. THE INDICTMENT ALLEGES THAT HE'S BEEN INVOLVED IN A MULTI-DECADE SCHEME TO LAUNDER -- EXCUSE ME -- TO EVADE TAX ON BILLIONS OF DOLLARS OF INCOME, AND RECORDS INDICATE THAT IN THE LAST FEW YEARS, HE'S BEEN DRAWING A SALARY FROM THE COMPANY THAT HE RUNS OF AT LEAST $1 MILLION A MONTH. SO FINANCIAL RESOURCES ARE NOT A PROBLEM FOR MR. BROCKMAN. IF HE WERE TO CHOOSE TO LEAVE THE DISTRICT OR LEAVE THE JURISDICTION OF THE UNITED STATES, FINANCIALLY THAT WOULD POSE NO PROBLEM FOR HIM.

ALSO, HE HAS THE PHYSICAL MEANS TO TRAVEL IN THE FORM OF A JET. MR. BROCKMAN HAS ACCESS TO A BOMBARDIER GLOBAL EXPRESS 6000 JET, WHICH IS A PRIVATE PLANE CAPABLE OF TRANSATLANTIC TRAVEL. THAT JET IS HANGARED IN THE DEFENDANT'S HOMETOWN, IT'S MAINTAINED IN HOUSTON, AND HE HAS ACCESS TO IT. HE'S TRAVELED INTERNATIONALLY SEVERAL TIMES IN THE LAST FEW YEARS.

SO HE HAS THE MOTIVE TO FLEE IN THAT HE'S FACING A VERY SERIOUS CASE, AND HE HAS THE MEANS TO FLEE.

AND THE THIRD CATEGORY I'D LIKE TO DRAW THE COURT'S

ATTENTION TO IS PERHAPS THE ONE THAT DISTINGUISHES THIS FROM OTHER CASES INVOLVING WEALTHY DEFENDANTS, AND THAT IS THE DEFENDANT'S PROPENSITY FOR OBSTRUCTIVE CONDUCT.

HE'S BEEN CHARGED WITH SEVERAL COUNTS OF OBSTRUCTION OF JUSTICE. ESSENTIALLY EVIDENCE TAMPERING AND WITNESS TAMPERING ARE THE CHARGES. BUT THE ALLEGATIONS IN THE INDICTMENT UNDERLYING THOSE COUNTS ARE THAT HE CAUSED ONE OF HIS COMPATRIOTS TO PHYSICALLY DESTROY EVIDENCE THAT WAS GOING TO BE PERTINENT TO THE INVESTIGATION.

THERE ARE ALSO OTHER ALLEGATIONS IN THE INDICTMENT ABOUT OBSTRUCTIVE CONDUCT, INCLUDING USING A PROPRIETARY ENCRYPTED E-MAIL SYSTEM, KEEPING SUPPLIES OF SPECIAL PAPER SPECIFICALLY FOR BACKDATING DOCUMENTS, USING UNTRACEABLE VONAGE ACCOUNTS TO COMMUNICATE WITH NOMINEES IN ORDER TO AVOID DETENTION BY LAW ENFORCEMENT, AND INSTRUCTING A CO-CONSPIRATOR TO ATTEND A CONFERENCE ON MONEY LAUNDERING USING AN ASSUMED IDENTITY.

SO THIS IS A SERIOUS CASE. THE DEFENDANT HAS THE MEANS AND THE MOTIVE TO FLEE, AND HE HAS A DEMONSTRATED CAPACITY FOR ENGAGING IN OBSTRUCTIVE CONDUCT.

UNDER THE CIRCUMSTANCES, IT'S THE GOVERNMENT'S POSITION THAT AN UNSECURED BOND WOULD NOT REASONABLY ASSURE THAT HE APPEAR AT SUBSEQUENT PROCEEDINGS, AND WE RESPECTFULLY REQUEST THAT THE COURT IMPOSE THE TRAVEL RESTRICTIONS THAT WE'VE AGREED TO, AND ALSO ORDER THAT THE DEFENDANT POST A $1 MILLION SECURED BOND.

THE COURT: THANK YOU, MR. PITMAN.

WHEN YOU SAY A $1 MILLION SECURED BOND, ARE YOU LEAVING IT FLEXIBLE THAT IT CAN BE SECURED BY ANY PROPERTY OR BY CASH, OR IS THERE A PARTICULAR SECURITY THAT YOU HAVE IN MIND?

MR. PITMAN: OUR PREFERENCE WOULD BE A CASH BOND, YOUR HONOR, BECAUSE IT'S -- IN THE EVENT OF A DEFAULT OR FLIGHT, IT'S SO MUCH EASIER TO PROCESS.

BUT I THINK ANY BOND THAT HAS GENUINE SECURITY WOULD OPERATE TO ENSURE THAT THE DEFENDANT IS AT LEAST TAKING THE BOND INTO CONSIDERATION WHEN EVALUATING HIS OPTIONS.

THE COURT: ALL RIGHT. I'LL GET MR. STEPHENS' RESPONSE, BUT I'LL JUST NOTE THAT AT THIS POINT IN THE PROCEEDINGS, I HAVE NO INFORMATION ABOUT MR. BROCKMAN BESIDES WHAT'S IN THE INDICTMENT, AND ORDINARILY THERE'S AN OPPORTUNITY FOR PRETRIAL SERVICES TO CREATE A REPORT FOR THE COURT THAT PROVIDES INFORMATION ABOUT THINGS SUCH AS HIS BACKGROUND, HIS FINANCIAL POSITION, AND FROM THAT REPORT THAT GIVES ME INFORMATION WHICH CAN GUIDE ME TO THE RIGHT ANSWER.

SO I'M HEARING FROM BOTH PARTIES THIS INFORMATION FOR THE FIRST TIME WITHOUT HAVING, YOU KNOW, MY OWN INVESTIGATOR. I DON'T HAVE MY OWN INVESTIGATOR TO FIGURE OUT THE ANSWERS TO THESE THINGS. I HAVE TO RELY UPON THE PARTIES AND PRETRIAL SERVICES TO PROVIDE THE INFORMATION TO THE COURT.

I TAKE IT, MR. PITMAN, THAT THERE'S NO CRIMINAL RECORD FOR MR. BROCKMAN. YOU WOULD HAVE MENTIONED THAT IF THAT WAS A

BASIS FOR YOUR REQUEST.

MR. PITMAN: YOUR ASSUMPTION IS CORRECT, YOUR HONOR. THERE'S NO CRIMINAL HISTORY THAT WE'RE AWARE OF.

THE COURT: ALL RIGHT. AND ALSO, I'LL JUST MAKE A NOTE THAT AFTER I -- THE PARTIES ARE IN AGREEMENT THAT MR. BROCKMAN IS GOING TO BE RELEASED, SO I'M IN AGREEMENT, TOO. I'M GOING TO BE ORDERING HIM TO BE UNDER SUPERVISION WITH SOME CONDITIONS.

I CAN HAVE PRETRIAL GIVE US A POST-RELEASE REPORT WITH ADDITIONAL INFORMATION THAT CAN MODIFY THESE CONDITIONS. FOR EXAMPLE, IF THE AMOUNT OF THE BOND IS TOO HIGH OR TOO LOW, PRETRIAL CAN TELL ME, AFTER THEY'VE DONE SOME INQUIRY, THAT IT'S TOO HIGH OR TOO LOW AND WE CAN THEN CHANGE THE CONDITIONS.

I'M JUST GOING TO DO SOMETHING TODAY BASED ON WHAT YOU TELL ME, BUT SOME DEEPER INQUIRY COULD BE APPROPRIATE.

MR. STEPHENS, BACK TO YOU TO GIVE ME FURTHER ARGUMENT ABOUT THE FINANCIAL CONDITIONS FOR RELEASE.

MR. STEPHENS: THANK YOU, YOUR HONOR.

AS TO THAT, IT'S OUR POSITION THAT THIS SHOULD BE A NO CASH BAIL SITUATION. MR. BROCKMAN BEFORE YOU HAS HAD AN EXCEPTIONALLY STABLE HOME LIFE, A WORK LIFE, AND A COMMUNITY LIFE, AND I'LL DETAIL THAT FOR YOU HERE SHORTLY, YOUR HONOR.

MR. BROCKMAN IS 79 YEARS OLD. HE HAS AND IS BATTLING SOME SERIOUS MEDICAL ISSUES, WHICH INCLUDE PARKINSON'S AND A HEART CONDITION, AND IN PAST YEARS HE HAS ALSO HAD TWO SEPARATE BOUTS

WITH CANCER.

AS COUNSEL HAS ACKNOWLEDGED, HE HAS NO CRIMINAL HISTORY. HE IS A FORMER UNITED STATES MARINE SERVING IN THE LATE 1950S THROUGH THE MID-1960S.

AS FAR AS HIS HOME LIFE, HE'S BEEN MARRIED FOR 50 YEARS TO HIS WIFE, DOROTHY. THEY RESIDE IN TEXAS AND IN COLORADO.

AS FAR AS HIS WORK HISTORY, YOUR HONOR, HE WAS -- HE HAD A GREAT ACADEMIC CAREER AT THE UNIVERSITY OF FLORIDA. THAT LED TO HIS FIRST JOB AT FORD MOTOR COMPANY. AFTER A FEW YEARS THERE, HE MOVED ON TO IBM AND HAS ALWAYS BEEN FOCUSSED IN THE SALES INTO THE AUTOMOTIVE INDUSTRY.

AND AFTER ABOUT FIVE YEARS AT IBM, IN 1970 HE STARTED HIS OWN COMPANY, A COMPANY CALLED UCS, WHICH IS UNIVERSAL COMPUTER SERVICES, AND IT WAS A SOFTWARE COMPANY, YOUR HONOR, THAT DEVELOPED SOFTWARE THAT CAR DEALERSHIPS COULD USE TO TRACK THE INVENTORY OF PARTS LOCATED WITHIN THE DEALERSHIP. THAT COMPANY, YEARS LATER IN 2006, MERGED WITH REYNOLDS. MR. BROCKMAN IS CURRENTLY THE CEO AND CHAIRMAN OF THE BOARD OF REYNOLDS CORPORATION.

HE HAS ALSO SERVED ON NUMEROUS COMMUNITY BOARDS, MOSTLY IN THE HOUSTON AREA. HE SERVES ON THE BOARD OF THE CANCER CENTER AT MD ANDERSON IN HOUSTON. HE IS ON TWO BOARDS AT RICE UNIVERSITY. HE'S ON THE BOARD OF TRUSTEES FOR THE UNIVERSITY, AND HE'S ON THE BOARD OF OVERSEERS FOR THE BUSINESS SCHOOL AT RICE.

HE IS ALSO ON THE BOARD OF TRUSTEES AT BAYLOR MEDICAL UNIVERSITY, AND AS WELL AS THE FORMER CHAIRMAN OF THE BOARD OF TRUSTEES AND CURRENTLY A LIFE TRUSTEE OF CENTRE COLLEGE IN KENTUCKY.

TO COUNSEL'S POINT, YOUR HONOR, ON RISK OF FLIGHT, MR. BROCKMAN HAS KNOWN ABOUT THIS INVESTIGATION FOR FOUR YEARS. HE'S HERE TODAY, YOU KNOW, VOLUNTARILY AFTER ACCEPTING A SUMMONS FROM THE GOVERNMENT.

HE IS NOT A FLIGHT RISK. HE'S CERTAINLY NOT A DANGER TO THE COMMUNITY. HE'S DEALING WITH VERY SERIOUS MEDICAL ISSUES, AND IT'S OUR VIEW THAT RELEASE ON HIS OWN RECOGNIZANCE IS APPROPRIATE, OR IF THE COURT WANTS TO FASHION SOME TYPE OF PERSONAL SURETY BOND, A PERSONAL SURETY BOND UP TO IN THE NEIGHBORHOOD OF $250,000 IS MORE THAN APPROPRIATE TO ASSURE HIS ATTENDANCE AT ALL THE COURT PROCEEDINGS.

THE COURT: AND GIVEN MY COMMENTS THAT I KNOW NOTHING ABOUT, YOU KNOW, HIS FINANCIAL SITUATION BEYOND WHAT YOU'RE PROFFERING TO ME AND WHAT THE GOVERNMENT IS PROFFERING TO ME, IT MAKES IT CHALLENGING FOR ME TO ESTABLISH THE AMOUNT THAT WOULD BE APPROPRIATE TO ENCOURAGE HIM TO COME TO COURT WHEN ORDERED, AND OF COURSE COMING TO COURT RIGHT NOW MEANS COMING REMOTELY AS HE'S DONE TODAY.

DO YOU HAVE A PROFFER AS TO, BETWEEN THIS ON HIS OWN RECOGNIZANCE OR 250 OR A MILLION DOLLARS AS TO, YOU KNOW, WHY I WOULD PICK THE LOWER NUMBER AND NOT THE HIGHER NUMBER?

MR. STEPHENS: YEAH. I JUST THINK A CASH BAIL NUMBER, YOUR HONOR, IS INAPPROPRIATE HERE. A PERSONAL RECOGNIZANCE BOND WILL ASSURE HIS APPEARANCE, AND PARTICULARLY GIVEN HIS ROOTS IN THE COMMUNITY, HIS ACTIVE SERVICE ON THESE BOARDS, THE LENGTH OF TIME THAT HE HAS BEEN IN THE COMMUNITY, WE DON'T SEE THE FLIGHT RISK GIVEN THAT HE'S KNOWN THAT THIS INVESTIGATION HAS BEEN ONGOING.

SO A LOT OF WHAT COUNSEL WAS TALKING ABOUT WAS WHAT HAPPENS IF CONVICTED, BUT WE DENY AND REFUTE ALL THE ALLEGATIONS AND ARE LOOKING FORWARD TO LITIGATING THIS.

THE COURT: AND DOES HE OWN PROPERTY THAT COULD BE POSTED AS A SECURED ASSET TO SECURE THE COMBINATION OF CONDITIONS?

MR. STEPHENS: YES, YOUR HONOR, I DO THINK THAT WE COULD HAVE SOME PROPERTY. I DON'T KNOW THAT IT'S ACTUALLY APPROPRIATE OR --

THE COURT: I KNOW YOU DON'T AGREE WITH IT, BUT IF HE DIDN'T OWN PROPERTY, THEN I WOULDN'T BE -- I WOULDN'T BE SUGGESTING THAT IT BE POSTED AS SECURITY, AND SOME DEFENDANTS DON'T OWN PROPERTY THAT THEY CAN POST AS SECURITY. IF HE DOES, THEN THAT BECOMES A POTENTIAL, A POTENTIAL ITEM THAT CAN BE POSTED TO HELP SECURE HIS RELEASE.

LET ME ASK OFFICER CHRISTIAN, WHO'S BEEN LISTENING IN, IF HE HAS ANY FEEDBACK ABOUT THE CONDITIONS OF SUPERVISION THAT HE WOULD ASSERT.

OFFICER CHRISTIAN: HELLO, YOUR HONOR, AND ALL PARTIES.

SO I DIDN'T GET THE INFORMATION REGARDING THE TRAVEL RESTRICTIONS. THERE WERE A FEW DISTRICTS LISTED, AND I HAD SOUTHERN DISTRICT OF TEXAS, COLORADO, NORTHERN DISTRICT, SOUTHERN DISTRICT OF NEW YORK, EASTERN DISTRICT, SOUTHERN DISTRICT OF OHIO AND NORTHERN DISTRICT OF OHIO.

WAS THERE A DISTRICT I MISSED IN THERE?

THE COURT: D.C. ALSO, DISTRICT OF COLUMBIA.

MR. STEPHENS, WAS THERE ANOTHER?

MR. STEPHENS: JUST THE NORTHERN DISTRICT OF CALIFORNIA WHICH I THINK MR. CHRISTIAN PICKED UP.

THE COURT: YEAH. THANK YOU.

I MAY HAVE INTERRUPTED YOU. GO AHEAD, OFFICER CHRISTIAN.

OFFICER CHRISTIAN: THE ONLY OTHER THING WOULD BE IF CONDITIONS WERE FOUND TODAY THAT HE WOULD BE RELEASED, WHICH IT SOUNDS LIKE THAT'S THE DIRECTION PEOPLE ARE HEADING, WE WOULD ASK THAT THE DEFENDANT CONTACT OUR OFFICE IN SAN FRANCISCO TOMORROW SO THAT WE CAN BEGIN PREPARING A PACKET FOR -- SINCE WE ARE THE CHARGING DISTRICT, A PACKET FOR WHAT WOULD BE THE SOUTHERN DISTRICT OF TEXAS, I BELIEVE, WHERE HOUSTON IS.

MR. PITMAN: YES.

OFFICER CHRISTIAN: SO WE WOULD BE PREPARING THAT PACKET FOR THEM.

BUT SINCE WE'RE THE CHARGING DISTRICT, WE HAVE TO START

WITH OUR DISTRICT, SO WE WOULD BE THE ONE WRITING THE REPORT AND ASKING THE SOUTHERN DISTRICT TO, YOU KNOW, FOLLOW THE CONDITIONS THAT ARE OUTLINED HERE OR MODIFY THEM HERE.

AND THAT ALSO WOULD BE THE CASE AROUND WHERE TO REPORT IN TEXAS FOR THE U.S. MARSHALS SERVICE. I'M UNCERTAIN WHERE TO TELL HIM TO REPORT, SO THAT WOULD BE SOMETHING WE WOULD NEED TO FIGURE OUT AND HAVE MORE INFORMATION FOR HIM TOMORROW.

MR. VARNADO: AND YOUR HONOR, THIS IS MR. VARNADO.

WE WILL ABSOLUTELY DO THAT AND FACILITATE THAT COMMUNICATION, MR. CHRISTIAN, AT WHATEVER TIME THE COURT DEEMS APPROPRIATE.

AND ON THE DISTRICTS, I WOULD JUST WANT TO REVISIT, BUT WE ACTUALLY THINK THE CONTINENTAL U.S. IS APPROPRIATE FOR THIS RESTRICTION. THERE'S NO REASON TO BE MORE RESTRICTIVE THAN THAT.

WE AGREED TO THE DISTRICTS SPECIFICALLY WITH THE GOVERNMENT IN HOPES THAT THEY WOULD MODIFY THEIR POSITION ON REQUIRING A CASH BOND OF A MILLION DOLLARS WHICH WE THINK IS INAPPROPRIATE.

SO IF THE COURT IS SO INCLINED AND IT'S EASIER, THEN CERTAINLY WE ASSERT THE CONTINENTAL U.S. AS TRAVEL IS FAIR.

THE COURT: THANK YOU, MR. VARNADO.

MR. PITMAN, MY REACTION TO THE NINE DISTRICTS -- I'VE NEVER HAD A CASE WHERE I PERMITTED TRAVEL TO NINE DISTRICTS BEFORE. MAYBE YOU'RE GOING TO TELL ME THIS IS THE BIGGEST TAX

CASE EVER, SO IT'S JUSTIFIED TO BE A LOT OF DISTRICTS.

BUT ADMINISTRATIVELY TO ENFORCE THAT FOR PRETRIAL, IT MEANS THEY'RE GOING TO BE TRACKING TRAVEL WITH -- YOU KNOW, THERE ARE PRETTY FINE LINES BETWEEN DIFFERENT DISTRICTS IN OHIO, AND IT INCREASES A CHANCE FOR AN ACCIDENTAL -- IF THE BOMBARDIER LANDS AT THE WRONG AIRPORT, NOW HE'S GOING TO BE ARRESTED. THAT'S A LOT OF SUPERVISION AND ENFORCEMENT WHEN, IF THE REAL CONCERN IS HE'S GOING TO LEAVE THE UNITED STATES, THEN WHY NOT JUST HAVE IT BE A RESTRICTION THAT HE NOT LEAVE THE UNITED STATES?

MR. VARNADO: AND, YOUR HONOR, I DO HAVE THE PASSPORT, JUST TO REITERATE THAT. I'M HOLDING IT HERE AND WE'LL TURN IT IN TO PRETRIAL (INDICATING).

THE COURT: THANK YOU.

MR. PITMAN: YOUR HONOR, I UNDERSTAND THE LOGISTICAL CONCERNS THAT YOU'RE RAISING. IN A TYPICAL WHITE COLLAR CASE LIKE THIS, WE WOULD SEEK TO RESTRAIN THE DEFENDANT TO THE NORTHERN DISTRICT AND ANY DISTRICT THAT HE NEEDED TO TRAVEL TO FOR HIS OWN DEFENSE.

I UNDERSTAND THAT THESE CONDITIONS MAY CREATE ISSUES FOR PRETRIAL, AS WELL AS FOR THE PARTIES, AND OBVIOUSLY IF THERE IS EXTRA TRAVEL THAT'S NECESSARY, MY ANTICIPATION IS THAT WE'LL BE ABLE TO WORK TOGETHER TO MAKE SURE THAT THOSE ARE STIPULATED AND THAT TRAVEL IS PERMITTED AS APPROPRIATE.

BUT AT LEAST, ESPECIALLY DURING THE INITIAL PART OF THE

CASE, YOUR HONOR, UNTIL THINGS GET SETTLED DOWN, WE WOULD LIKE PRETRIAL AND THE U.S. ATTORNEY'S OFFICE TO BE INVOLVED IN UNDERSTANDING MR. BROCKMAN'S TRAVEL PATTERNS.

WE WERE PERHAPS TOO GENEROUS IN AGREEING THAT, YOU KNOW, GIVEN THE FACT THAT HE'S REPRESENTED BY ATTORNEYS ALL OVER THE COUNTRY, THAT WE WOULDN'T DISPUTE THAT HE SHOULD TRAVEL TO EVERY DISTRICT NECESSARY TO FACILITATE VISITS WITH THOSE LAWYERS, BUT THAT IS THE -- THAT'S WHY THERE ARE SO MANY AGREED DISTRICTS.

TYPICALLY IT WOULD BE THE NORTHERN DISTRICT AND WHEREVER THE LAWYER IS, AND IN THIS CASE THERE ARE MANY OF THEM.

THERE'S -- THE EXCEPTIONS, OF COURSE, ARE THE DISTRICTS IN OHIO, AND WE JUST THINK IT'S APPROPRIATE TO ALLOW HIM TO CONTINUE TO TRAVEL THERE IF NECESSARY IN THAT HE IS THE CEO OF A COMPANY THAT EMPLOYS THOUSANDS OF PEOPLE.

OFFICER CHRISTIAN: AND IF I COULD ALSO CLARIFY, YOUR HONOR, WOULD HE BE RESIDING IN HOUSTON? IT SOUNDED LIKE HE MIGHT HAVE A FEW PLACES HE'S RESIDING, WHICH MAKES SUPERVISION CHALLENGING. SO WHEN THE ORDER OF CONDITIONS OF RELEASE ARE STIPULATED TODAY, HAVING THE ADDRESS THAT HE WILL REMAIN AT IS MORE HELPFUL FOR SUPERVISION, AND USUALLY IN THIS CASE WHAT WE WOULD REQUEST.

MR. VARNADO: AND HE WILL BE RESIDING IN HOUSTON, YOUR HONOR. HE MAY PERIODICALLY TRAVEL TO COLORADO, BUT HE WILL BE RESIDING IN HOUSTON.

OFFICER CHRISTIAN: UNDERSTOOD.

THE COURT: ALL RIGHT. I'M JUST REVIEWING THE PROPOSED CONDITIONS. ONE MOMENT.

(PAUSE IN PROCEEDINGS.)

MR. VARNADO: YOUR HONOR, I DO HATE TO INTERRUPT. I DID WANT TO JUST MENTION ONE MORE THING.

AGAIN, APOLOGIES. MR. VARNADO HERE.

WHEN WE HAD SPOKEN WITH THE GOVERNMENT -- I JUST HEARD MR. PITMAN SAY THE STANDARD CONDITIONS, AND I'M NOT LOOKING AT THE SAME FORM THAT YOU ARE, BUT WE SPECIFICALLY HAD THAT THE GOVERNMENT WAS NOT SEEKING A CONDITION RELATED TO FIREARMS. THAT MIGHT BE IN THE STANDARD FORM OF THE NORTHERN DISTRICT, I'LL DEFER TO MY COLLEAGUE, MR. STEPHENS. BUT THAT WAS ONE IN PARTICULAR THAT THE GOVERNMENT SAID THEY WERE NOT -- DID NOT HAVE AN INTEREST IN TRYING TO ENFORCE.

THE COURT: YEAH, HE DID NOT READ THAT ONE AS A CONDITION THAT HE WAS PROPOSING.

MR. VARNADO: VERY WELL.

THE COURT: IT WAS MR. STEPHENS WHO READ IT, BUT MR. PITMAN AGREED TO THE CONDITIONS THAT MR. STEPHENS READ.

ALL RIGHT. I'M THINKING -- WHEN DO THE PARTIES WANT TO APPEAR BEFORE JUDGE ALSUP? ASSUMING I'M GOING TO ISSUE RELEASE CONDITIONS HERE, WHEN DO YOU WANT TO HAVE THAT NEXT APPEARANCE?

MR. STEPHENS: HOW ABOUT THE FIRST WEEK OF NOVEMBER, YOUR HONOR? OR SECOND WEEK?

THE CLERK: ACCORDING TO HIS CALENDAR, NOVEMBER AVAILABILITY IS NOVEMBER 17TH OR 24TH.

MR. STEPHENS: SO EITHER ONE OF THOSE DATES IS GOOD BY ME, BY US.

MR. VARNADO: MAYBE THE 17TH WOULD BE PREFERABLE, YOUR HONOR.

THE COURT: ALL RIGHT. WE'LL SET IT NOVEMBER 17TH BEFORE JUDGE ALSUP IN SAN FRANCISCO. THAT'LL BE A REMOTE HEARING I ASSUME, UNLESS HE ORDERS OTHERWISE.

WHAT TIME IS THAT ON THE 17TH?

THE CLERK: 2:00 O'CLOCK, YOUR HONOR.

THE COURT: 2:00 O'CLOCK PACIFIC TIME, NOVEMBER 17TH BEFORE JUDGE ALSUP.

AND WOULD THE PARTIES LIKE TO EXCLUDE TIME UNTIL THAT DATE FOR SPEEDY TRIAL?

MR. STEPHENS: THAT'S FINE FOR ADEQUATE PREPARATION.

THE COURT: ALL RIGHT.

MR. BROCKMAN, WHAT WE'RE TALKING ABOUT IS YOU HAVE A RIGHT TO A TRIAL WITHIN 70 DAYS OF BEING CHARGED. THAT TIME PERIOD CAN BE EXTENDED SO THAT YOUR ATTORNEYS CAN INVESTIGATE THE CHARGES AGAINST YOU AND COMMUNICATE WITH THE GOVERNMENT AND LOOK AT ANY DISCOVERY THAT THE GOVERNMENT PRODUCES TO THEM.

SO FOR THE EFFECTIVE PREPARATION OF COUNSEL, I'LL EXCLUDE TIME UNTIL NOVEMBER 17TH, 2020.

MR. STEPHENS: YOUR HONOR, WE WOULD MAKE OUR REQUEST

FOR RULE 16 DISCOVERY.

THE COURT: MR. PITMAN HEARS THAT AND WILL RESPOND.

MR. PITMAN: YES.

THE COURT: VERY GOOD. ALL RIGHT.

THESE THEN WILL BE THE CONDITIONS OF RELEASE WHICH I'M ORDERING, AND I'M GOING TO REVIEW THEM WITH YOU AND PROVIDE YOU A WRITTEN COPY. MY DEPUTY IS GOING TO ASSIST ME BY WRITING THEM DOWN AS I RECOUNT THEM, AND WE'LL GET IT TO YOU LATER TODAY.

THESE ARE GOING TO BE TEMPORARY CONDITIONS BECAUSE I WANT PRETRIAL TO LOOK INTO THESE CONDITIONS FURTHER. THEY MAY AGREE THAT THESE ARE THE PERMANENT ONES, BUT IF THEY HAVE PROPOSED MODIFICATIONS, I WANT TO SET A ZOOM HEARING IN TWO WEEKS FROM TODAY ON THE 29TH AT WHICH POINT WE CAN COME BACK TO MODIFY THESE, IF APPROPRIATE, OR YOU MAY ALL AGREE THAT THEY'RE FINE AND DON'T NEED MODIFICATION. BUT I DO WANT TO HAVE AN OPPORTUNITY TO FOLLOW UP ONCE I'VE GOTTEN SOME MORE FINANCIAL INFORMATION ABOUT MR. BROCKMAN.

THE TEMPORARY CONDITIONS ARE THAT MR. BROCKMAN IS ORDERED RELEASED ON A $1 MILLION UNSECURED BOND. AND IT MAY VERY WELL BE THAT THERE ARE SOME PROPERTIES THAT COULD BE POSTED TO SECURE THAT, AND I WANT PRETRIAL TO LOOK INTO WHETHER THERE'S AN APPROPRIATE PROPERTY OR PROPERTIES THAT CAN SECURE THE BOND, BUT I'M USING THAT FIGURE. IT'S, OF COURSE, MORE THAN WHAT THE DEFENSE SUGGESTS, AND IT'S NOT SECURED LIKE THE GOVERNMENT

WOULD BE REQUESTING, BUT IT'S IN LIGHT OF THE FACT THAT MR. BROCKMAN HAS KNOWN ABOUT THE POTENTIAL CHARGE FOR A NUMBER OF YEARS AND HAS NOT FLED.

ON THE OTHER HAND, THERE ARE SERIOUS CHARGES OF CONCEALMENT AND EVASION AND EVIDENCE TAMPERING, DESTRUCTION OF EVIDENCE, THAT ARE CONCERNING AND SUGGEST, WITH MR. BROCKMAN'S FINANCIAL ABILITY AND CAPABILITY TO FLEE THE COUNTRY, THAT THERE SHOULD BE A SIGNIFICANT BOND. SO THAT'S WHY I'M THINKING A MILLION DOLLARS.

IT COULD BE THAT PRETRIAL SAYS THAT THAT SHOULD BE LOWERED OR RAISED OR SECURED IN SOME WAY, BUT THIS WILL ALLOW PRETRIAL TO GATHER MORE INFORMATION FROM THE PARTIES AND MAKE A REPORT TO THE COURT.

AND ON THAT $1 MILLION UNSECURED BOND, THE DEFENDANT MUST COME TO COURT WHEN ORDERED. THE NEXT COURT DATE IS BEFORE ME ON THE 29TH OF OCTOBER FOR A FOLLOW-UP.

AND IF HE IS SENTENCED, HE MUST SURRENDER TO SERVE ANY SENTENCE IMPOSED.

WHILE ON RELEASE, HE MAY NOT COMMIT ANY FEDERAL, STATE, OR LOCAL CRIME.

HE MUST NOT HARASS, THREATEN, INTIMIDATE, INJURE, TAMPER WITH, OR RETALIATE AGAINST ANY WITNESS, VICTIM, JUDGE, OR JUROR, AND MAY NOT OBSTRUCT ANY CRIMINAL INVESTIGATION.

HE'LL BE SUBJECT TO PRETRIAL SERVICES' SUPERVISION, AND HE MUST CONTACT PRETRIAL BY TOMORROW -- OFFICER CHRISTIAN WILL

PROVIDE THE NUMBER FOR THAT FOLLOW-UP COMMUNICATION -- AND REPORT THEREAFTER TO PRETRIAL SERVICES.

I EXPECT THAT THE HOUSTON OFFICE WILL BE DOING THE LOCAL SUPERVISION, BUT PRETRIAL WILL ESTABLISH THAT.

THE PASSPORT I'VE SEEN IN COUNSEL'S HAND. THAT MUST BE SURRENDERED TO PRETRIAL SERVICES.

AND OFFICER CHRISTIAN, WHEN SHOULD WE MAKE THAT DEADLINE?

OFFICER CHRISTIAN: IF WE CAN MAKE THAT DEADLINE MONDAY SO THAT THEY CAN OVERNIGHT IT TOMORROW?

THE COURT: ALL RIGHT. SO TO PRETRIAL BY MONDAY, THE 19TH.

AND MR. BROCKMAN MAY NOT APPLY FOR A PASSPORT OR OTHER DOCUMENTS TO LEAVE THE UNITED STATES.

WHILE ON RELEASE, MR. BROCKMAN MAY NOT CHANGE HIS RESIDENCE OR TELEPHONE NUMBER WITHOUT ADVANCE APPROVAL OF PRETRIAL SERVICES. THAT MEANS HE NEEDS TO PROVIDE PRETRIAL WITH HIS PRIMARY RESIDENCE AND TELEPHONE CONTACT INFORMATION, AND HE MAY NOT CHANGE THOSE WITHOUT ADVANCE APPROVAL OF PRETRIAL SERVICES.

I WILL IMPOSE THE TRAVEL RESTRICTIONS THE PARTIES AGREED TO. I AM CONCERNED THAT THEY ARE ADMINISTRATIVELY CHALLENGING TO ENFORCE, AND PRETRIAL CAN LOOK INTO WHETHER THESE REMAIN TO BE VIABLE OR NOT.

BUT THE CONDITIONS OF TRAVEL ARE RESTRICTED TO THE SOUTHERN DISTRICT OF TEXAS; THE DISTRICT OF COLORADO; THE

NORTHERN DISTRICT OF CALIFORNIA, THAT'S THIS DISTRICT; THE SOUTHERN DISTRICT OF NEW YORK; THE DISTRICT OF COLUMBIA; THE EASTERN DISTRICT OF VIRGINIA; AND THE SOUTHERN AND NORTHERN DISTRICTS OF OHIO.

AND MR. PITMAN IS OBLIGATED TO PROVIDE TO THE DEFENSE INFORMATION ABOUT WHERE THOSE DISTRICTS ARE SO THAT MR. BROCKMAN CAN COMPLY. WE'VE GOT A MAP OF THE NORTHERN DISTRICT OF CALIFORNIA. THAT'S FROM MONTEREY UP TO THE OREGON BORDER. BUT SOME OF THOSE OTHER DISTRICTS ARE GERRYMANDERED AND CAN BE A BIT TRICKY ABOUT WHERE THEY ARE.

SO, MR. PITMAN, IF YOU CAN ALERT THE DEFENSE TO WHERE THOSE JURISDICTIONS ARE, WHERE IT'S NOT AN ENTIRE STATE -- FOR EXAMPLE, THE SOUTHERN DISTRICT OF TEXAS HAS GOT SORT OF JAGGED LINES -- PLEASE PROVIDE THAT INFORMATION TO THE DEFENSE SO THAT MR. BROCKMAN DOES NOT HAVE AN INADVERTENT VIOLATION GOING SOMEWHERE WHERE HE'S NOT SUPPOSED TO.

MR. PITMAN: YES, YOUR HONOR.

THE COURT: MR. PITMAN, DID I HIT THE STANDARD CONDITIONS THAT YOU WANTED TO BE IN THERE?

MR. PITMAN: YOU DID, YOUR HONOR.

THE ONLY ADDITIONAL WRINKLE DURING THESE COVID TIMES IS THAT I WOULD LIKE THE COURT TO ORDER MR. BROCKMAN TO REPORT TO THE MARSHALS, AS WELL AS PRETRIAL, IN THE SOUTHERN DISTRICT OF TEXAS FOR BOOKING.

THE COURT: THANK YOU FOR THAT REMINDER, AND I AGREE.

I DON'T KNOW HOW THE MARSHALS IN HOUSTON ARE DOING THEIR BOOKING PROCESS. IT'S DIFFERENT IN EACH JURISDICTION IN THE COUNTRY.

SO WHAT I'LL ASK YOU TO DO IS TO PROVIDE MR. STEPHENS WITH THE INFORMATION FOR THE U.S. MARSHALS IN HOUSTON.

AND MR. BROCKMAN, I'M ORDERING YOU, WITHIN THE NEXT WEEK, BY THE 22ND -- AND YOUR ATTORNEYS CAN ESCORT YOU FOR THIS VISIT -- TO BE BOOKED BY THE U.S. MARSHALS THERE, AND THAT'S SO THAT -- YOU WON'T BE GOING INTO THEIR CUSTODY, BUT IT'S SO THAT YOU'LL BE FINGERPRINTED, I EXPECT, AND WILL BE -- IT'LL BE FORMALIZED THAT YOU HAVE BEEN PRESENTED WITH THESE CHARGES AND ARE IN THE SYSTEM. THAT'LL HELP SECURE YOUR FUTURE COURT APPEARANCES.

MR. VARNADO: YOUR HONOR, JUST SO THE COURT KNOWS, WE DID REACH OUT TO THE MARSHALS SERVICE ALREADY. THINGS ARE WORKING RATHER STRANGELY. I'M GOING TO GO DOWN THERE TOMORROW TO FIGURE OUT WHAT TO DO.

BUT THANK YOU FOR THE WEEK TO DO THAT. I DIDN'T WANT TO TRY TO COMMIT TO DOING IT TOMORROW BECAUSE THINGS ARE OPERATING RATHER STRANGELY. WE'LL GET HIM PROCESSED.

THE COURT: VERY GOOD. AND IF THE ANSWER IS THAT THEY CAN'T ACCOMMODATE YOU, YOU LET ME KNOW. BUT I HOPE THAT WITHIN A WEEK'S TIME YOU CAN MAKE THAT HAPPEN SAFELY, BUT IT IS A PART OF THE PROCESS THAT DOES NEED SOME PERSONAL PARTICIPATION.

ALL RIGHT. MR. BROCKMAN, IF YOU FAIL TO COMPLY WITH THESE CONDITIONS, THE CONSEQUENCE IS THE GOVERNMENT COULD SEEK A $1 MILLION FORFEITURE FROM YOU. ALSO, YOU COULD BE HELD IN CUSTODY FOR THE REMAINDER OF THE CASE.

IF YOU COMMIT A CRIME WHILE ON RELEASE, THE PUNISHMENT IS INCREASED FROM THE ORDINARY STATUTORY MAXIMUM.

MR. BROCKMAN, DO YOU UNDERSTAND THE CONDITIONS OF RELEASE WHICH I DESCRIBED?

THE DEFENDANT: YES, I DO.

THE COURT: AND DO YOU AGREE TO FOLLOW EACH OF THEM?

THE DEFENDANT: I DO.

THE COURT: MAY I SIGN ON YOUR BEHALF THE RELEASE ORDER?

THE DEFENDANT: YES.

THE COURT: ALL RIGHT. I'LL DO THAT. WE'LL SEND YOU A WRITTEN COPY OF IT.

AND, AGAIN, WE'RE GOING TO COME BACK IN TWO WEEKS BY VIDEO. SO AT THAT TIME, ANY PARTY CAN TELL ME IF WE SHOULD MODIFY THOSE TEMPORARY RELEASE CONDITIONS, OR YOU CAN TELL ME BEFORE THE HEARING THAT YOU DON'T SEEK MODIFICATION AND THEN WE'LL JUST VACATE THE APPEARANCE ON THE 29TH.

OFFICER CHRISTIAN: IF I COULD ALSO PROVIDE THE ADDRESS TO OVERNIGHT THE PASSPORT AND THE PHONE NUMBER?

MR. STEPHENS: THAT WAS MY NEXT QUESTION, JAY.

OFFICER CHRISTIAN: OKAY. WE'RE ON THE SAME PAGE

THEN.

SO IT'S GOING TO BE 450 GOLDEN GATE AVENUE, AND THAT'S GOING TO BE SUITE 18-5497. AGAIN, IT'S 450 GOLDEN GATE, SUITE 18-5497, AND IT CAN BE ADDRESSED TO PRETRIAL SERVICES. AND THAT'S IN SAN FRANCISCO, CALIFORNIA, 94102.

AND PROVIDING THE CONTACT INFORMATION FOR THE DEFENDANT TO FOLLOW-UP TOMORROW AFTER 10:30 FOR THE ASSIGNED OFFICER TO ARRANGE THE INTERVIEWS, AND TO ALSO HOPEFULLY HAVE SOME LOGISTICS FIGURED OUT ON OUR END, THAT NUMBER IS 415-436-7501. 415-436-7501.

MR. STEPHENS: VERY GOOD. THANK YOU.

OFFICER CHRISTIAN: YEAH.

THE COURT: ALL RIGHT. I THINK THAT'S EVERYTHING. WE'VE EXCLUDED TIME, WE'VE SET THE FURTHER COURT DATES, AND PRETRIAL WILL BE COMMUNICATING FURTHER WITH YOU ABOUT ANY FURTHER MODIFICATION OF THOSE RELEASE CONDITIONS.

MR. PITMAN, ANYTHING FURTHER WE SHOULD DO IN THIS CASE?

MR. PITMAN: NO. THANK YOU, YOUR HONOR.

THE COURT: THANK YOU VERY MUCH.

MR. STEPHENS, ANYTHING FURTHER?

MR. STEPHENS: NO. THANK YOU, YOUR HONOR, FOR TAKING SO MUCH TIME ON A VERY CROWDED CALENDAR.

THE COURT: YOU'RE WELCOME. THANKS ALL FOR YOUR WAITING AND PATIENCE.

THAT CONCLUDES THIS MATTER.

HAVE A GOOD DAY, MR. BROCKMAN. WE'LL SEE YOU IN TWO WEEKS.

OFFICER CHRISTIAN: I THINK MS. HARRELL HAS ONE EXTRA QUESTION.

THE COURT: OH, YES.

THE CLERK: IF COUNSEL CAN PROVIDE THE DEFENDANT'S ADDRESS VIA E-MAIL SO I CAN PUT IT ON THE BOND, THAT WOULD BE VERY APPRECIATED.

MR. STEPHENS: YES, MS. HARRELL. WE'LL DO THAT RIGHT AFTER THIS CALL.

THE CLERK: THANK YOU.

THE COURT: ALL RIGHT. THANK YOU VERY MUCH.

MR. PITMAN: THANK YOU, YOUR HONOR.

THE COURT: AND WE'LL GET THE RELEASE ORDER OUT TO YOU LATER TODAY.

MR. STEPHENS: THANK YOU, YOUR HONOR.

THE COURT: ALL RIGHT. HAVE A GREAT DAY. THANK YOU VERY MUCH.

THE DEFENDANT: BYE. THANK YOU.

(THE PROCEEDINGS WERE CONCLUDED AT 12:59 P.M.)

CERTIFICATE OF REPORTER

I, THE UNDERSIGNED OFFICIAL COURT REPORTER OF THE UNITED STATES DISTRICT COURT FOR THE NORTHERN DISTRICT OF CALIFORNIA, 280 SOUTH FIRST STREET, SAN JOSE, CALIFORNIA, DO HEREBY CERTIFY:

THAT THE FOREGOING TRANSCRIPT, CERTIFICATE INCLUSIVE, IS A CORRECT TRANSCRIPT FROM THE RECORD OF ZOOM PROCEEDINGS IN THE ABOVE-ENTITLED MATTER.

Lee-Anne Shortridge

LEE-ANNE SHORTRIDGE, CSR, CRR
CERTIFICATE NUMBER 9595

DATED: OCTOBER 27, 2020